REFLECTIONS

ON THE

NATURE OF INFLAMMATION,

AND

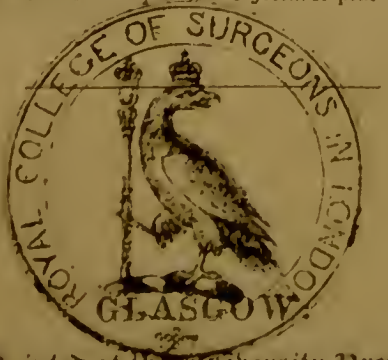
ITS ALLEGED CONSEQUENCES.

By DAVID BADHAM, M.D.,

ONE OF DR. RADCLIFFE'S TRAVELLING FELLOWS FROM THE UNIVERSITY OF OXFORD.

"Ουκ οιμαι γαρ τινα ουτως ευτυχη γραφην ηγεισθαι, η μηδεις αντειρει 'αλλ' εκεινην ευλογον νομιστεον, η μηδεις ευλογως αντειρει."—CLEMENS ALEX.

"Il me semble que, dans toute science qui ne réleve pas immédiatement de l'application mathématique, il y a deux études a faire; la première est celle des faits demontrés, et des idées de détail ou de generalization, qui en sont la consequence; la seconde consisté à porter son attention sur une serie *d'idées qui ne sont encore que des conjectures* plus ou moins fondées—des matériaux *en réserve*."



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TO

SIR HENRY HALFORD, Bart., M.D., F.R.S.,

&c. &c. &c.

PRESIDENT OF THE ROYAL COLLEGE OF PHYSICIANS,

THE FOLLOWING PAGES ARE,

WITH GREAT RESPECT,

INSCRIBED BY

THE AUTHOR.

Rome, *1st January*, 1834.

Glasgow College, 25*th Jan.* 1834.

TO THE READER.

Since it would be an act of very questionable prudence in me, from my relation to the author of the following pages, to prefix to them any preface, or observations of mine, and since it will be inferred, of course, that I fully approved of their publication, I would merely say, that having accepted the responsible duties of Editor, without the possibility of subsequent conference with the author, I request the reader to bear that disadvantage in mind in any passage that may seem to require it.

Charles Badham.

THE

NATURE OF INFLAMMATION, &c.

As the object which an author may propose to himself in the publication of his opinions respecting any subject connected with our profession, are, perhaps, better stated at once, I think it right to apprize the reader that mine, in the following pages, is, first, to consider the value of those arguments which have been advanced in support of the doctrine of *inflammatory products;* and, after having canvassed these arguments, to inquire further respecting the nature of inflammation itself, and the probable origin of some at least of the various results assigned to it—my intention, however, being by no means that of compiling a circumstantial history of doctrines or opinions, but rather of submitting to the competent reader whatever there may be of truth or consistency in my own reflections.

The older pathologists, after they had traced up the processes of suppuration, gangrene and ulcer into previous inflammation, exacted no further services from an agent which modern schools have made nearly omnipotent; but as the study of morbid anatomy advanced, and the knowledge of various disorganizations accumulated, it was not long before the *hardening* and *softening* of structures, together with a great variety of other changes,

of which the different organs of the body were found susceptible, in colour, texture and consistence, came to be also considered as results of a "travail inflammatoire," sometimes mitigated into the more specious doctrine of *irritation*, but at others hardily asserted, and at present, with little reserve, adopted in France. Now, if the word irritation, or irritability, simply expressed the fact of organic actions being called into play by an unknown stimulus, it would scarcely be obnoxious to criticism; it is the employment of this word, as the followers of Brown employ it, to designate an hypothetical agent that cannot be permitted. Chomel, one of them, is guilty of this abuse, when, in order to explain the origin of morbid products, or the nature of those actions which conduct to them, he thinks it enough to use the word *irritation*, and then to regard this irritation, itself a fiction, as the sufficient cause of almost all the phenomena which pathology investigates; Coutanceau, another expositor of the same doctrine, would remind us, to the same purpose, "that the disorganizations, known by various names, are not things, or entities, but the mere consequence of chronic *irritation*;" and we read in Billiard, a yet more avowed Broussaist, that "inflammation is the grand agent of *all disorganization*, and, that without violating truth, or permitting ourselves to be seduced by forced analogy, we may affirm, that by its agency more changes of tissue are effected than by the united influences of external and mechanical causes, or the whole of those which escape observation!" Accordingly, we find all these authors, as they proceed, conferring upon this agent the power of producing, not only various, but even *opposite* results. When thus abused, indeed, it signifies little whether they designate such an agent by the term *inflammation* or *irritation*, for inflammation or irritation are indifferently abstractions of our own minds; and, being such, equally incapable of *explaining* those phenomena *said* to result from them. "Car on ne doit jamais prendre des principes ou maximes *abstraits*," says

Condillac, "pour des principes propres à mèner à *des decouvertes.*" Yet, what has not been explained by one or other of these terms, from the formation of pus to the production of hydatids and worms! There was doubtless a time, but it has long since passed away, when far less assuming dogmas respecting inflammation would have been received with hesitation and distrust. but, "les suppositions, d'abord arbitraires, sont devenues incontestables par l'addresse avec laquelle on les a employées."

Before we engage to *explain* complicated phenomena, on any common principle, we should be in a condition to establish their connexion with each other; but the author of the "Phlegmasies Chroniques" pursues a bolder course than most of his disciples, assumes postulates for proofs, and clears away all difficulties by the omnipotence of a word. This is the peculiar fault of all framers of systems, who *write* as if they really conceived themselves to be imparting solid information, when in fact they are but calling new words into existence, or giving currency to phrases, or collocations of words, which seldom fail to mystify those who do not reflect, and to perplex those who do. It were difficult to conceive, under the pretence of extreme accuracy, a greater want of it (though the writings of several French writers furnish equally conspicuous examples) than the dictum of Broussais, that *all* those adventitious masses which may develop themselves in the various organs, nay, that even *fever* itself is but the result of a *latent* and local inflammation; though neither of these qualifying terms, could we permit ourselves to adopt it, would put us in possession of any sounder view on the subjects to which he would apply them. Well, indeed, may we say with the philosopher quoted above, as we turn over the never-ending pages of the new pathology, "Comment quand le *principe* est vague *l'expression* aura-t-elle quelque precision?"

There are one or two points, however, to be briefly considered before we can proceed even to that first part

of our subject, in which I would attempt to prove, that not even one of the supposed results of inflammation, far less the whole of them, can be philosophically attributed to this obtrusive agent. The results themselves, as it is acknowledged on all sides, most widely differ; but then it is said, in order to remove the objection from that diversity, that they only differ as the efficient differs in *activity;* or, only differ, as others are pleased to say, according to the *texture* which is the seat of the inflammation; or, if these two considerations be still held inadequate, according to the *constitution* or *idiosyncrasy* of the individual affected; to all which expedients some would add, in order to the removal of all possible difficulty, according to the *exciting cause*, which as they apprehend may be either an *ordinary*, or a *specific* and *peculiar* stimulus. but I would fain attempt to show that inflammation is always the same *action* whether acute or chronic in form or duration, by whatever cause produced, and *in* whatever tissue or modification of circumstances existing.

1. It is needless to remark how very much we are apt to be ruled by words, when it is obvious that such qualifying terms, as chronic and acute, leave *inflammation*, the *thing* or *action qualified*, identically the same, as indeed a remarkably clear writer on this subject does not hesitate to admit. "There is only a difference between the two, of degree, (the chronic being marked by a period of less violent disturbance,) but it is the *same kind* of disturbance; for we find that the acute forms of inflammation may pass into the chronic, and the chronic may relapse into the acute." Either of these facts is familiar enough; Mr. Laurence, however, adds another, which goes in further proof of the identity of inflammation, as to its *nature*, under *all* circumstances, viz., that *chronic* inflammation does not necessarily imply a *slow* maturation of abscess, nor is a *tardy* suppuration incompatible with an inflammation to which the name of chronic could *not* be given.

2. To consider different, and dissimilar morbid changes, as produceable by one inflammatory action,

merely modified by the structure of parts, and, therefore, leading to different results, exhibits, I think, a want of clearness in our original conceptions about inflammation at all, or even the instruments it employs. For is not this, in the first place, to forget *that the action of inflammation is carried on in vessels*, (*originating* at least, as I hope hereafter to show, in the red capillaries only,) and consequently should not be affected by the nature of the *parenchyma* or *tissue*, of which the various parts of the body are made up, and *through which* the inflamed vessels merely *ramify*. Hence, I am not disposed to accede to the following passage which occurs in one of Mr. Laurence's lectures:—"When you are informed that inflammation is an augmented action of the vessels of a part; when you learn that the seat of inflammation is in the capillaries; when you know, at the same time, that the arrangement of these vessels presents a difference in each organ, you will naturally be prepared to conclude that the inflammatory process will have its peculiar characters in each structure; that there will be something particular that will distinguish the process in every part in which it occurs." On the contrary, I should be disposed to contend that when we consider that inflammation is an augmented action of the vessels of a part; when we see reason for fixing such action in the *red* capillaries, and have, at the same time, no possible reason for supposing that there is any difference in the arrangement of these vessels in different organs, we are naturally prepared to conclude that the simple inflammatory process will have *no* peculiarity of character in different structures; and that there will be nothing specific to distinguish this process in whatever part it may occur. Besides which objection, conclusive as it already seems, many *different* disorganizations may occupy *different portions at once* of one and the *same* inflamed tissue. Now, *identity of cause* cannot produce *diversity of effect;* diversity of effect is produced; the cause then cannot be the same, nor, under such circumstances, can we safely attribute

any *one in particular* of these results to the hypothetical cause of the *whole* of them; for, can we ever predict which of these products, or whether any of them will take place till it has actually occurred?

3. Nor is there much meaning in the employment of such otherwise qualified expressions as suppurative inflammation, gangrenous inflammation, ulcerative inflammation, since this is to forget that we may have "one inflammation attended with suppuration and gangrene in one part of the same tissue, and resolution in another;" a fact from which Mr. Laurence has inferred "that one species of inflammation is susceptible of more than one result," but from which I would, on the contrary, infer the incorrectness of dividing one action, like inflammation, into any *species* at all.

Inflammation is generally considered by pathologists as a *morbid* action assumed by the vessels of a part. This morbid action, then, might issue in a morbid result; but not surely in more than *one* result, or else the action ceases to be one, and becomes several actions; and when two or more morbid results are referred to inflammation, it is clear that they cannot all be parts or consequences of that process, unless they are parts of each other. But inflammation, as we shall see, is a *natural action in kind*, and, therefore, cannot conduct to any *unnatural result*. that it *is* a *natural* action in kind, appears from the circumstance, that parts the most violently phlogosed, by no means necessarily become disorganized, (which, therefore, when they do, we must refer to some *other* action connected with the inflammatory one); and, also, because in many organic changes accompanied by inflammation, the *travail inflammatoire* sometimes does not appear till organic change is far advanced, and sometimes not even till the conclusion of that organic change. Now, in thus mistaking the *contingent* for the *essential*, and mixing up the phenomena of phlogosis, always the same, with some other morbid action which *may accompany* it, but which certainly forms no part of it, the confusion of our general discourse on this subject is easily foreseen.

For if it be impossible to refer *all* the supposed results of inflammation to that *one* action, which inflammation must necessarily be, (even though the word be taken to express a *new*, that is, a *morbid* action imposed on the part,) it will be not less so, to refer *any* of these results at all to inflammation, if, as I think, it is only an augmented *natural* action of a part. But let us, with Mr. Laurence, consider it as a vital action altered in mode; will this obviate the difficulty of tracing morbid productions, or other pathological changes to this source? and *can any* conceivable alteration in the *mode* of vital action become the *contingent* efficient, as some would make it, of every possible disorganization of tissue? now, to enter on this subject somewhat in detail, let us first inquire whether even *suppuration*, the most familiar of all changes, can claim to be a direct consequence of inflammation, and not rather the result of some added and peculiar mode of action, which may either be associated with the inflammatory one, or operate independently of it.

As the statements employed by writers on this subject are not correct, the inductions derived are necessarily faulty. When, for instance, Hunter, Laurence, and other pathologists, whose names contribute to the reception of their opinions, tell us that inflammation leads to the formation of pus, in consequence of being carried *so high* that the state of parts, on which such inflammation has been exercised, is destroyed, and so, that these parts lose the inflammatory action, and acquire another which fits them for producing matter, it is surely reasonable to inquire how, if this explanation be just, the suppurative process should so often *fail* to occur in many intense cases of highly inflammatory diseases; how it happens that the most confirmed inflammations, seated even in the *cellular membrane*, (that tissue in which most disorganizations appear to commence, and which, above all others, most readily inclines to the suppurative action,) should ever fail to terminate in this way, which that they *do*, the experience of many, besides Dupuyt-

ren, sufficiently establishes—but, if I am right in my conjectures, that *simple* inflammatory action cannot, whatever be its intensity or duration, put the inflamed parts, to use the words of Hunter, "in a way to form pus," such anomalous cases disappear.

We have many positive reasons besides this negative one, (the fact of intense inflammation often not followed by suppuration,) for supposing the perfect independency, so to speak, of these two processes, and none, perhaps, greater than the utter want of proportion between the amount of a given inflammation, and the amount of the future purulent secretion; a result so indefinite, as should alone, one would think, suggest that it is rather the *remote* cause of a given inflammation that determines the *extent* of its supposed consequence: just as the diseased spleen, so frequently an attendant upon ague, is "not caused *by* the intermittent, but by the *cause* of the intermittent, since its appearance *at all* does not depend on the violence of the ague, nor on its duration; and since the amount of it, when it *does* occur, does not depend on the gravity of the disease with which it is so frequently connected." (Elliotson.) A sentiment somewhat analogous had, however, long before been implied by Aretæus, who refers enlarged spleen, without any mention of intermittent fever, to *marshy ground.**

But, it may be said, that if inflammation and suppuration depend upon the *same* efficient, and yet are independent of one another, why does the former affection so very *commonly* introduce the other? and does not the *order* in which they occur point to inflammation as the *cause* and suppuration as the *effect?*

I shall have occasion presently to notice, that suppuration may occur where no inflammation can be shown to have proceeded; and even were this general

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* "Χωρια ελκωδεα, ὑδατα πλατεα, αλμυρα βρωμωδεα ωρων, το Θηριωδες μετοπωρον." Hippocrates had also said, that to those who drink paludal or stagnant waters, "σπληνας αει μεγαλους ειναι."

order of antecedence and consequence admitted to be universal, it would not therefore make one the efficient of the other, for *both* may be as readily results of some separate cause, as be connected with each other in the relation of cause and effect; nay, it is just as easy to suppose the two tendencies to inflame and to suppurate, to be simultaneously developed, each independently of the other, as to suppose that one introduces the other; for, as we cannot tell under what law of the economy inflammation itself is developed, we may equally submit to acknowledge our ignorance of that disposition of parts, or that agency upon them, which determines suppuration. It may even be supposed, and from analogy it is not very unlikely, that when, in conformity to some occult principle of the economy, inflammation is set up, this may, so far from producing, even *prevent*, during the time that it prevails, that formation of matter, which may in possibility *not* be able to manifest itself till such inflammation *decline*. May we not also urge, on our side of the argument, the inaptitude of the same part to exhibit two different actions at once? thus the germs of two of the exanthemata, measles and scarlatina, may both be received into the system together, but show themselves in the order of their respective incubation; and the very same *may* be true of suppuration and inflammation, for the production of which one and the same *remote* cause may evidently suffice. Still further, the act of suppuration is justly held to resemble a *secreting* process, and on this very account pus ought to be looked upon with great hesitation as an *inflammatory product*. We know nothing, indeed, of the *actions* which constitute secretion; we cannot even presume whether they are the *same* actions in the instance of each secreted product, nor at present perhaps dare we even hope to arrive at the laws of that vital chemistry which must lend its aid to the mechanism of structure in giving origin to all new products. We do not know how it is brought about, (to use the words of an eminent surgeon,) "that the capillary vessels of one part

deposit the substance of muscle; those of another, the substance of bone, and so on," but though the present state of knowledge afford no positive conclusions on these heads, yet we are all agreed in never supposing an action of the *nature of inflammation* in *any* such cases. Moreover, we see inflammation followed by a suspension of the secerning action when *natural*, by an *arrest* to the further secretion of its fluid in a gland or membrane thus affected, and, hence, we derive the strong argument from analogy, that suppuration is not an inflammatory process nor pus an inflammatory product, and that inflammation has no more to do with forming pus than it has to do with forming urine; "*ici* seulement c'est une fonction normale, *la*, c'est une fonction pathologique liée à une disposition nouvelle dans l'organe qui en est le siège."

In further distrust of the doctrine which asserts the connexion of cause and consequence between inflammation and suppuration, which by the way is a very ancient one,* I would now argue,

1. That suppuration may exist *without* having been preceded by inflammation. 2. That if inflammation *did* always precede the suppurative process, even this would not assure the connexion of the two.

To pass over, with a simple reminiscence, the very frequent occurrence of high local inflammation unattended by suppuration, (though a very formidable objection in itself to the current opinion is to be found in that familiar fact,) for the opposite case of suppuration without inflammation, I have only to refer the reader to authorities which go back at least as far as de Häen. This proposition may, I am well aware, be addressed to a reluctant reader; yet, besides, the respectable names that may be quoted in support of it, I have myself, in several instances, had undeniable evidence of pus formed without any discernable preceding inflam-

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* "ην δε ηδε (φλεγμονη) εις γεννησιν ηκε πυου. And again, αιτιη δε φλεγμασιη." Aretæus.

mation; instances where tumours formed on various parts of the body, had, from this very absence of inflammation, been considered as *adipose* tumours requiring extirpation, till, on the introduction of a lancet, an awkward mistake was just averted by the flow of variable quantities of pus from the punctured orifice. Nay, I presume this mistake to be not so very unfamiliar to surgeons; for purulent collections unaccompanied by inflammation have been too frequently recorded as having occurred in the parenchyma of organs to admit of any rational scepticism on this point. Cases are mentioned by authors, and they are by no means of very unfrequent occurrence, in which the brain, the liver, the spleen and the kidnies have become the seats of purulent collections, though not only the autopsy has discovered no trace of previous inflammation, (which I believe to be the universal fact, when these parts are the seats of purulency,) but no ambiguous evidence could even be extorted from *symptoms*, of the probability of such a state.

Andral, an author, than whom few can pretend to more *accuracy* in pathological research, has declared himself *familiar* with instances in which almost every organ of the body has become the seat of purulent formations *without* previous phlogosis; and he mentions one very remarkable case in which vast abscesses occurred at once, in *many* different parts of the body, some pointing externally, when the patient suddenly began to void with his urine a white, thick, puriform matter, after which he got well. Here a purulent *constitutional* action is plainly seen to begin and end without inflammation. Twice, in la Charité, in one year, it also occurred to find purulent matter included within a *clot of blood* in one of the cavities of the heart, this organ being in every respect healthy, and never having afforded any trace of disease. Dr. P. M. Latham, in the really valuable lectures on the heart with which he has favoured the profession, has noticed a similar appearance, though we do not learn in his case whether the previous symptoms were those of inflammation. In such in-

stances one might find also some foundation for the hypothesis of de Häen concerning the existence of pus intermingled with the blood, and of its occasional elimination from this fluid in the formation of abscess; but as the accuracy or inaccuracy of this opinion has nothing whatever to do with my argument, I shall, for the present, make no further remark upon it.

Abernethy tells us that he has found abscesses containing genuine pus in the middle of adipose-sarcomatous tumours; and as these tumours are held by him to be wholly *uninflammatory* in their origin and progress, the purulent secretion which they contain cannot be ascribed to such origin.

Reynaud records instances of pus diffused through coagula of blood in the *healthy* spleen; and Roux, whose experience cannot be contested, expresses his familiarity with facts of the same nature in very unequivocal terms. "Que de fois, à l'ouverture de cadavres, on decouvre des abscés, dont on n'avait pas même *soupconné* l'existence!" the citation of other consentient testimony may be spared; it would be needless to multiply examples, for if the authorities I have quoted cannot, as they surely cannot, be gainsayed, they are already enough for those who are open to the reception of evidence; while further statements would scarcely make one proselyte from among those who are sceptical by *habit*, and believers by education, in *latent* local inflammation. My own conclusion, from these and similar cases, does not, however, rest here, I conclude, further, not merely that suppuration *need not* have an inflammatory origin, but that a genuine suppurative process *cannot* have its origin in a purely inflammatory cause. If suppuration may occur in even a single instance without inflammation, why not in *all* instances? one efficient being in all instances adequate to one effect, to invoke two, has been always held contrary to sound philosophy. "Ισορροπος γαρ η φυσις, ως εν ισορροπω," (Aretæus.) But I foresee that an objection may be taken to cases, confessedly not common, and acknowledge that reasoning from exceptions to the general

course of things may be deemed inconclusive; but I would remark that *exceptions* in grammar, for instance, or other *imperfect* work of human ingenuity, are not of the same *kind* with what we are accustomed to call such in physics: nature has no real exception; physiology and pathology admit of no real anomalies—we must not, therefore, under this obnoxious title, cast out of our account any undoubted *facts* that occur in medical investigation, but rather study to reconcile them with, than oppose them to, what we call *principles;* remembering always, (to use the language of De Candolle,) "That these are to be regarded as *experiments* which nature is performing for our instruction."

Before proceeding to the second proposition, that the mere precedency and succession of phenomena, are not absolute proofs of the relationship of cause and consequence, I should desire (even supposing inflammation and suppuration to observe this invariable order of occurrence) to remove from our pathology, Hunter's ill-judged division of suppuration into one kind, of *inflammatory* origin, and another which is, he says, unattended by over-excited blood-vessels. His assigned characters of the two processes, and of their results, are seldom so considerable, and sometimes so trivial, that it would be difficult for any but a determined theorist not to look upon them as mere modifications of one and the self-same action. Dr. Thomson, in his interesting volume on inflammation, tells us that he has frequently found no difference between the products of *inflammatory,* and what the French, are in the habit of calling "*cold* abscess." And Abernethy has accurately detailed *many cases* of lumbar abscess, where, *inflammatory* symptoms having been entirely *wanting*, the *pure, concrete, laudable,* or *inflammatory* pus of Hunter, was copiously poured forth on puncture. We cannot, therefore, take the existence of even *laudable pus* after death as a proof of *preceding phlogosis.* Such cases also show that suppuration is but one action, which, becoming under certain circumstances

slightly modified, gives rise to corresponding modifications of one common result. Now, let us look at the case of what *is* really held to be inflammatory suppuration; "here the fluid *first* deposited," says Mr. Laurence, "has a serous, and sometimes a bloody character, though afterwards we may find in the same part a secretion, having all the characteristics that belong to pus;" is not the difference here stated between the bloody serum first effused, and the genuine pus, or subsequent product, at least as *great* as *any* conceivable difference in the qualities of any two kinds of dissimilar pus, let them differ ever so much? yet, in the first case, no one pretends that *two essentially different* actions are concerned, the one for the production of *serous*, the other for that of *purulent* fluid: the same remark, then, might surely apply to all *differences in purulent secretions* which are ascribable to different modifications of but *one* action. Does not the very division of abscesses, into chronic and acute, *make* them consequences of one action; otherwise how could they run so imperceptibly into each other? such, accordingly, is Roux's division; his abces *aigus* and abces *chroniques* run into each other, while his third order is to be called *acute*, if we consider the time taken for their development merely, but *chronic* (froids) if we look to the absence of antecedent inflammation. Now, a common cause, a common action, must needs be supposed, where effects are found to run so imperceptibly into each other. Dupuytren saw the necessity of some such admission, and therefore holds "*all* abscess to be of the same nature." On *that* point I agree with him perfectly, but wholly differ with him in the conclusions which he has laboured to deduce. "L'ancienne division des abces," says this writer, "en abces chauds ou inflammatoires, et abces *froids*, considerée comme s'appliquant à des tumeurs de nature différente, et provenant des actions vitales *dissemblables*, doit être *abandonné*." This I believe to be true, but not so what follows: "Les tumeurs purulentes ont toutes la même origine—

l'inflammation; celle çi ne manque dans aucun cas, mais elle peut affecter des tissus différentes, présenter des nuances d'intensité variables à l'infini, et selon les circonstances les collections purulentes se forment avec lenteur ou rapidité, acquièrent des dimensions considerables ou restreintes, et renferment des liquides dont les qualités diffèrent singulièrement." We are also told, *more majorum*, by this writer, that though the fact of inflammation is *proved* by the fact of suppuration, yet the inflammation may have been *latent* during life. Now, we do know something of latent *heat* in unorganized bodies, but inflammation is not *heat*, it is a *vital action*, and of a vital action of which we have had no evidence during life, we may well be disposed to call the existence in question afterwards, even though the *pretended* effect of such action be pointed out. It is surely the more *philosophical* course, of the two, to hold that abscess *never* results from inflammation than to insist that it *always does;* for that absence of symptoms which may not unfairly be urged in support of the *heretical* opinion, leaves us in hopeless confusion if we are determined to be *orthodox*. And here the general question might properly come in, whether *symptoms* or *post mortem* appearances are, in pathology, the *most* to be relied on, in cases where they would lead to *different* conclusions respecting the existence or absence of a *travail inflammatoire;* but, is it not sounder doctrine to hold, that in all cases, where the symptoms during life have been *said* to be at variance with the *post mortem* phenomena, that the evidence of the former ought to be esteemed as greatly *surpassing that* furnished by the other? or rather, is it not true, that *that* other, furnishes, by itself, no evidence of *any* action, unless we shall have well studied, and by study intimately connected it with such action in the *living* body? can the detection of any organic lesion, be used in *argument*, unless in conjunction with previous *symptoms? we* should never place an unreal *antagonism* between two things that must be connected, a morbid action and a morbid result, and one object of this paper is to

maintain that we virtually do so, when we make suppuration *dependent* on inflammation ; for so long as we will insist on an hypothetical efficient in order to the accomplishment of this process, we are wandering far away from that course which might lead to a more adequate interpretation of the laws by which it is governed ; we retard the progress of pathology by all assumptions which by their *convenience* conceal our real ignorance, and thus distract us from the rigorous pursuit of more extended observation. Can too much regard be paid to symptoms ? Shall we relapse into that absurd dogma of Brown as to these being comparatively valueless ? symptoms may not be infallible, but neither are our senses infallible—so far from it, that we frequently employ these senses to correct each other. Symptoms, it *must* be admitted, are the *best* evidence afforded us, and as probability in various matters, a probability which seldom issues in certainty, is all we can ever have to guide us in morals, or the business of life, we must be content with it in medicine. Dr. Bright, in his excellent work on diseases of the brain, has cautiously made the remark, "that the *results* of a continued irritation on this organ may be *sometimes* shown" leaving the inference that the existence of this irritation must chiefly, if not altogether, be traced in the *symptoms* which it produces. And I remark, in one of Dr. Elliotson's lectures, a hint, which admits of universal application, "not to consider, in dropsy, the state of the urine as our guide to venesection, but to consider whether the general *symptoms* are those of inflammation or not;" these authors, then, should scarcely disagree with me, that the appearance of an abscess after death, which had been *unattended* by inflammatory *symptoms* during life, was, in fact, rather an evidence *against* inflammation having existed in the site of such abscess. In *slow* organic disease the changes that *do* occur are slow, and the *symptoms* are, therefore, ill-defined, and not unlikely to be overlooked. In such cases *post mortem* appearances are all we have to guide us ; but the reverse

of all this obtains in *inflammation*, which is essentially an *acute* affection, and where its effects on the living body can alone be trusted.

In tracing the progress of medicine we are continually involved in the confusion arising from a too exclusive reference, either to appearances in the dead body, or the expression of suffering, or of functional disturbance (*symptoms*) in the living. Yet when I contend for the greatly superior value of the *latter* in medical investigation, it is not, however, without being aware that the *same* symptoms probably often, and sometimes of necessity, attend *dissimilar* affections of the same organ; but with this admission, I had rather place my reliance on symptoms, with all their fallacies, than with Dr. Gooch consent to undervalue at once both *these*, and the morbid results exposed by anatomy to the eye, in favour of an evidence which appears to me *far* more ambiguous and deceptive; the *effect of remedies*. This remarkably clear-headed and otherwise sound writer, seems, in particular, disposed to pay far too little regard to *symptoms*, "which," he says, "can never do more than suggest probabilities, while *trial of remedies* is the *only* conclusive proof." To such an inference I cannot bring my mind, nor join the writer in his censure of Dr. Armstrong for having too largely insisted upon symptoms, (taken, of course, together with *post mortem* appearances,) in settling the *kind of disease;* nor yet agree with him, that the effect of remedies constitutes not only an essential, but the *most important* part of the history of a disease, other evidence being of no value but as it throws light upon this; for whence do we else derive the *experience* that must first justify the very employment or experiment of the remedies we administer? is not this *entirely founded* upon a *careful comparison* of symptoms which must first have been noticed, with those now actually present? take an inflammatory affection: how do we know that it is inflammatory? is it because, as Dr. Gooch would tell us, it yields to *antiphlogistic treatment*, or because

it manifests a known set of symptoms, and because the autopsy is seen to agree with what these symptoms have, in former cases, been found to intimate? how did we come to adopt this treatment? is not the very epithet it enjoys derived from the success with which it had been found to combat certain admitted symptoms of phlogosis? it is symptoms, then, that *give origin* to treatment, and hence it follows, that any knowledge of the supposed *nature of disease*, founded upon the effect of *treatment*, is so far from conclusive, that it *derives its* whole value *from* symptoms. As to morbid appearances alone, take Angina Pectoris, and say of what value are the *infinitely various* appearances which have presented themselves on dissection. In relation to Dr. Gooch's argument in favour of his opinion respecting the superior *logical* value of the effects of treatment, and the insufficiency of symptoms and dissections to correct diagnosis, namely, that the same treatment will produce opposite results when symptoms are quite identical; first, I doubt it exceedingly; but then, I cannot doubt our *inaccuracy* and the *imperfect* exercise of our observation, and so, am at no difficulty to conceive how it may happen, that two diseases, apparently similar, that is, which present strong points of analogy, will require a various administration of remedies. Besides, as most diseases are *compound* affections, we may have noticed only those symptoms which are the most pronounced; *such* symptoms may, indeed, be similar in two *prima facie* similar cases, and yet involve essential differences, concealed under less marked manifestations. Dr. Gooch himself teaches, that there are two kinds of *peritonitis;* one inflammatory, one not. I agree with him, or rather am so taught by him. But are we to make this fact out by *symptoms* or by *treatment?* I should say, by the first, because I hold the symptoms of inflammation, rigorously ascertained, to be always the *same*, and that the value of treatment in the interpretation of disease will be in exact proportion to the *accuracy* with which symptoms have first been studied. Of the symp-

toms of inflammation, perhaps, pain may be the least certainly *essential*, while *redness*, heat, and intumescence, must almost necessarily go together, and so become unambiguous. Pain, almost always present in inflammation, may yet be *neurotic;* pain, too, commonly appears *before* the other symptoms, that is, in fact, before there is inflammation. Where inflammation, as it undoubtedly may, exists *without pain*, such existence may perhaps be inferred from the success of the *treatment;* but this admission involves no contradiction to what I have just been stating of the relative value of semeiology and of therapeutic, for an antiphlogistic treatment may be rationally instituted upon the evidence of other and less equivocal signs than pain, as in pure inflammatory fevers where there is no pain.

I come now to the *second consideration*, whether inflammation necessarily precedes the formation of matter. Let us first admit for argument's sake, as fact, that which, however, some reasons have been assigned for hesitating to believe, even if we adopt the *statement* of those who maintain this doctrine. Let it be conceded, that when suppuration occurs, it always *follows* inflammation; such concession will not yet establish the relationship of cause and consequence? Cartilage is not esteemed the efficient of bone, because the cartilaginous structure in some instances *precedes* the osseous, for some bones, we know, are formed without passing through the intermediate stage of cartilage, and some cartilages are destined to continue such through life. Many procedures might be quoted in the animal economy in which two occurrences are seen very closely *united*, and yet most certainly not in the relation of cause and effect. It is an admitted law in the economy of warm-blooded animals, that where "the animal temperature is found the highest, the muscular contractility will be likewise found the greatest." Can it be, therefore, maintained, that contractile action *depends* upon animal heat? the *apparent* relation between these two really unconnected facts is

satisfactorily resolved by the consideration, that *before* the animal temperature is dissipated after death, contractility has already ceased; and, as in this instance an accurate physiologist (Bostock) considers contractility as the "unknown efficient of familiar effects" I would consider suppuration as an effect of an *unknown* cause, which may be, indeed most frequently *is*, but not by necessity, conjoined with inflammation. Another instance of apparently connected, but in fact merely conjoined phenomena, may be found in the separation of carbonic acid from the blood, and the *change of colour* in that fluid as it passes into the pulmonary capillaries; phenomena so united in the process of respiration, as to have appeared to many in the erroneous relation of dependence one on the other, which would have been a precipitate, and an erroneous conclusion; for all blood, observes Coutanceau, that has formed a *secretion* of any kind, becomes *dark in consequence;* and as the carbonic acid and watery halitus of the lungs are now justly viewed in the light of secreted fluids, it follows that their separation cannot be the producing agent of the *light* colour of the blood. "La formation de l'acide carbonique et de la vapeur aqueuse d'une part, et de l'autre la coloration du sang veineux, n'*ont point une cause commune*, et doivent être considerées comme deux phenoménes indépendans, quoique survenues dans la même acte functionnel." The external world might be cited for abundant instances of coincidence without connexion, such as light without heat, and *vice versâ*, but these would be less available to my argument.

It is an excellent observation of Abernethy, "that it is absurd to assert we have no other knowledge of cause and effect than what results from the continued observation of the priority of the one, to the consequence of the other," and he shows, by several apt illustrations, how a *rational* assurance on this subject is obtained. "If," says he, "I am able, in the absence of the object, to represent upon paper, a spherical or many-

sided figure, do I not manifest a knowledge of the causes of light and shade beyond that which my perceptions alone would have produced? If also, I can at will present the angle of a prism to a luminous body, so as to produce the regular exhibition of the rainbow colours, do I not exhibit a *knowledge* of the cause of such effects?" Now, agreeing perfectly, as I do, in the justness of these remarks, I would say that if we could, by first producing inflammation, *secure* the appearance of suppuration, we should, indeed, exhibit a knowledge of the *cause* of this last phenomenon: but as we cannot insure this, the *cause* of the suppurative process may lie elsewhere; for to admit inflammation as the cause, because it often comes before, is the very error here alluded to, the error of supposing "that we have no knowledge of cause and effect beyond that which results from observation of the precedency of the one, and the succession of the other." If, again, we should adopt Marjolin's idea, (declining, however, his general and vague *theory* of inflammation,) that it is simply "an augmentation of vital force," an excess of action in a part, but still of *natural* action, we are quite at a loss to conceive that it can furnish any new products either in changes of structure or morbid secretion, such as the formation of pus.

It has been urged as argument in favour of suppuration being *produced* by inflammation, that the quality and consistence of a purulent secretion are various, as more or less inflammatory action has been present; but has it not also been ascertained, that at least an equal influence over the qualities of a purulent secretion is exercised by the natural processes of assimilation and digestion, and even by moral causes; and can these, then, lay claim to be considered as *efficients* of suppurative action? If *pus* changes its consistence and becomes thin in consequence of high inflammation existing in the parts around it, the urine in *nephritis*, or inflamed kidney, also becomes more limpid and watery; the rule appears to apply to all secreted products, that, on the accession of inflam-

mation, they are either arrested or rendered more thin and watery. The inflammatory stimulus seems first to *narrow*, perhaps, if it does not *hermetically seal*, the mouths of those vessels which are not destined to carry red blood; hence, perhaps, the substitution of a more limpid fluid for the original and grosser one.

The following passage from Ferrus,* seems to vacillate between the old opinion and certain doubts similar to those which I have ventured to state, and will appear, to those who consider it a little, as singularly inconclusive and unsatisfactory, as it does to me. At first I hoped to have found a confederate in an author who begins his treatise on nephritis by stating, that when inflammation has not proceeded as yet to some one of *those organic modes, which we are accustomed to hear called terminations*, it leaves no mark behind it by which its previous existence may be ascertained, but proceeds, "il en est de même *pour tous les organes parenchymateux*, le travail inflammatoire *peu developée* n'altère point leur contexture d'une *manière sensible*," in all which I *agree* with him, (and *disagree* with most other pathologists in consequence,) but far otherwise in the very next sentence of his paper, which is a relapse into the old doctrine, "Mais si au contraíre la nephrite est très-intense, elle peut amener la degenerescence du parenchyme de cet organe."

There is a remark of Guersent, which would be far more valuable had it been less partial and confined, of the application of which, however, to this question, I would rather not lose the advantage. It contemplates the *connexion* of *tubercle* with phlogosis. "Des que deux états peuvent se rencontrer *isolément* et *independamment* l'un de l'autre, rien ne s'oppose à ce qu'ils puissent se trouver *réunis* sans que l'un soit precisément ni la cause ni l'effet de l'autre."

And here I would willingly refer the reader to Condillac's clear, eloquent, and profound work, entitled,

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* Dict. des Sciences, Med. Art. Nephrite.

"Traité des Systemes." His chapter on the use and abuse of hypothesis in physics, cannot be too carefully studied, and might in several places be cited in support of the objections I have urged against the modern dogmas on inflammation; as, for instance, where he gives as the legitimate end of hypothesis, "pour rendre sensible certaines vérités que l'experience fait connaitre. But," he continues, "to do this, ce n'est pas assez qu'on n'en puisse pas démontrer l'impossibilité; elle sera défectueuse si on ne la concevoit point de tout; *car ce qui ne se conçoit pas ne sauroit contribuer à rendre sensible une vérité*," and as he justifies Newton in rejecting the Cartesian doctrine on the score, "qu'elle parut plus propre à multiplier les difficultés, qu' a expliquer les phenomenes," I would claim a similar indulgence from the unprejudiced, and on precisely the same grounds.

Can adhesion, any more than suppuration, be considered as indebted to previous inflammation for its *production?* Some previous effusion is implied, indeed, in all adhesion, and effusion is the result of inflammation; but, then, inflammation will not determine the *nature* of the effused fluid, and when it *happens* to be *coagulable lymph*, (the material out of which all adhesion is formed,) this is plainly independent of inflammation, and indebted to a specific action for its production. "The power of vessels in inflammation," says Hunter, "to throw out this coagulable lymph, is destroyed by a variety of circumstances, amongst others by a low state of the vital powers; therefore, in all cases where blood-vessels are *liable to become inflamed*, this state of the constitutional powers, owing to the risk of blood being vitiated, is to be guarded against in our treatment." So full an admission of the existence of phlogosis, without effusion of coagulable fluid, (the bond by which adhesion is effected,) points rather to a coincidence, than to any more intimate relation between the two.

In holding gangrene also to be independent of an in-

flammatory origin, though the onus of showing that its efficient, or one of its efficients, *is* inflammation should rest with those who maintain that opinion, I shall endeavour to meet the common-place notions in favour of what I conceive to be false doctrine, conducting, as most false doctrines in our profession are found to do, to occasional errors in practice. From the very loose mode of description adopted by most writers on medicine, gangrene would appear to consist merely in the *death* of a part; or, to adopt the language of Hebriard, "*extinction of the life of a part, with re-action of the vis conservatrix of the surrounding ones.*" Mr. Laurence, with his usual perspicuity, speaks of gangrene as consisting in the death of a part, *together with a peculiar* change of structure in that part, the result of a previous and peculiar vital action, and this so different from *that* death of a part which is involved in the death of the *whole system*, that some kinds of mortification are to be considered as complete preservatives against putrefaction." Hence, he infers the inutility of anti-septics in the treatment, because the change which occurs in mortification is "not to be considered as identified with putrefaction."

Gangrene, unlike suppuration, is *reputed* by most writers to have *several* efficients, such as intense cold; the infliction of any serious local injury; the direct interruption of a supply of blood to a part by pressure or ligature; diseases of the heart; and, lastly, inflammation. Marjolin, indeed, has gone beyond his countrymen, in assigning still more causes than these, (he reckons *ten*,) though I think all of them may be reduced to one of the above heads: indeed, correctly speaking, none of them should be quoted as direct agents for a state which I cannot help thinking must needs consist in some *common* condition, to which parts are *first* reduced by the variety of assigned causes, *before* they become gangrenous. Inflammation, by some reputed the *constant* precursor, by *all* as *one* of the efficients of gangrene, is only, I conceive, a *remote* and occasional cause of this

process; for if, on a superficial view of the subject, it appears that in violent inflammation the disorder in the circulation is carried sometimes to a pitch which the part is incapable of sustaining, so that the blood stagnates and the part perishes, (Laurence); yet at other times the inflammation preceding gangrene is, we know, so very inconsiderable (as, for example, where it succeeds to the application of a blister, or to scarifications made to let out the fluid of anasarca,) that nothing would be less philosophical than to attribute gangrene, in such case, to a "travail inflammatoire," to which it bears no sort of proportion. On the other hand, are we at all better prepared to explain the action of gangrene with Mr. Laurence, "by a comparison of the amount of action with the degree of *power* of a part?" for this statement supposes gangrene essentially to consist in excess of action over power, and precludes altogether the necessity of that *peculiar* action, which has been so properly insisted on by this able writer in his very definition of the process. Then, as to *debility*, *this* can neither *produce* gangrene or any other disease; for whether such debility be of the kind contemplated by Mr. Laurence, in which "action exceeds power," or be of that kind in which both action and power are diminished in an equal degree, it seems incapable of the work assigned to it If we turn our attention for a moment to the condition of hybernating animals, and the examples of debility afforded by parts of the animal economy that have become congealed, we may find instances in both, of great depressions in the energies of vital and organic life; yet, in the *first* example, gangrene never occurs, in the *second*, only when the frozen part is *imprudently* stimulated. Neither does gangrene necessarily occur in constitutions weakened by cachetic diseases, by scurvy, by valvular disease of the heart, or, in short, by any hopeless asthenic ailment; nor, lastly, does it occur by any *necessity*, but only by contingency, in those cases of intense inflammation where the balance of action and

power is wholly destroyed. But as cause and consequence are linked together by strict *necessity*, an avoidable or contingent consequence cannot logically be considered as a consequence at all. Parts, then, do not sphacelate in consequence of either excess or deficiency of their natural action, nor yet by a destruction, through inflammation, of the equilibrium, which, in health, is maintained between action and power. When such parts do sphacelate, it is in consequence of a *new* and *morbid* action being called into play, for which pathology wants a name, but which might be called *mortifactive*, since the word *mortification* implies a result, a passive state, and loses sight of the fact, that the part must have been *killed*. In short, some positive and peculiar action of a *destructive tendency*, (not merely an inflammatory, which is only an increased action,) must be supposed, before the "death of a part, with reaction of the vis conservatrix in the surrounding ones" can possibly take place. We can hardly, I think, continue to maintain the supposed relation of cause and consequence between the processes of inflammation and gangrene, when, on the one hand, we consider that parts largely endowed with the "nisus formativus," and more suited on that account to accept and sustain inflammatory action, as, for example, the muscular solid, are yet very *little* disposed to gangrene; and, on the other, that ligament, tendon, and bone, parts by no means equally capable of reproduction, or equally liable to inflammation, fall readily enough into that condition; or, lastly, if we reflect that it is in the depending extremities where the circulation is less vigorous, and in "*external* parts chiefly, and very rarely in the *internal* organs of the body," that gangrene generally occurs. (Laurence.)

If, then, neither an inflammatory, nor, by parity of reasoning, any other condition of the body, or of a part, which may chance to *precede* gangrene, can, in any instance, be considered as its *direct* or efficient cause, it remains to be inquired, secondly, of what nature such a general cause may be supposed to be. In the attempt

to ascertain this, we should consider what is the *state*, and what are the precise appearances presented by parts just previous to sloughing. Now, as inflammation very frequently does *precede* gangrene, it may not be irrelevant to consider whether the state of an *inflamed* part can throw any light upon the subject, or suggest any sense in which gangrene may be considered as brought about by *inflammation*. I own myself disposed to think that inflammation, together with all those other assigned states of parts said to produce gangrene, will be found to be merely remote causes, concurring only in *one* circumstance, and that one, a tendency to cause *congestion*, and by reason of such congestion, the ulterior or *mortifactive* action. Not to anticipate my own notion of the nature of *simple* inflammatory action, I shall here, and in relation to the present subject only, appeal to one or two of the phenomena presented by inflamed parts.

1. That in local inflammation an increased quantity of blood is sent to a part, is abundantly shown, I conceive, by the redness and general congestion of the tissues of such part, (Hunter, indeed, has placed this fact beyond dispute by actual experiment.)

2. More blood actually *circulates* through the part than in a state of health. This, indeed, has been *denied* by many, and though I believe it to be a certain fact, yet it is not from the arguments adduced by Mr. Laurence, which seem somewhat inconclusive. "If," says this distinguished observer, "there were merely a larger quantity of blood sent to a part, and it remained stagnant, you would not have the vessels distended, as you will find takes place in inflammation of the hand or forearm." Now, as it is quite certain that distention is not caused by *motion*, but by the quantity of material moved, the turgescence of vessels, mentioned by Mr. Laurence, cannot be the *consequence* of an *accelerated*, any more than it is of a *retarded*, circulation; turgescence can have no other cause than *redundancy*, and this would produce the phenomenon in question,

equally in motion or at rest. Again, Mr. Laurence would infer a greater *circulation* of blood through an inflamed part, from the circumstance of more of this fluid flowing out, and from a larger number of vessels yielding it, during the performance of a surgical operation on an inflamed, than on an uninflamed tissue. In a case where the hand and forearm were inflamed, he had an opening made in *each* arm, and found that, within the same space of time, dividing the veins at the same instant, about three times more blood flowed from the vessels of the inflamed limb than from those of the sound; but is there no room for an objector to inquire whether the more copious bleeding, which here followed the division of the inflamed vessels, might not have been owing rather to an increased development of *some* vessels, and even to the formation of *new* ones, than solely to an increased circulation of the blood through the inflamed parts? for when an "appel des fluides" takes place, the part must of *necessity* become more vascular in order to accommodate the increased supply of blood which it receives; it may also be conceived that more blood will *continue* to flow from the veins of an inflamed part, *because* the ordinary vessels, now enlarged, and a numerical increase of the smaller ones, must invite more blood to distend their respective calibres out of the circulating mass; as a series of pipes will derive from the main trunk which fills them, more or less, in proportion to their number, and to the *sum* of their respective capacities.

Not that, however, I am at all indisposed to the conclusion, that there is in fact a greater circulation generally in inflamed parts; only I think it is to be supported on surer grounds of evidence than those afforded by Mr. Laurence. I should come to the conclusions at which this eminent writer arrives, simply from the very familiar facts of the increased colour of the skin from its augmented heat,* from its exalted sensibility,

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* θερμη γαρ αιματος προκλησις. Aretæus.

all of these conditions being consequences of the more copious arrival of *arterial*, let it be recollected, not the delay of *venous* blood. Of course, where there is more arterial, there will *come* to be also more venous blood, as a necessary consequence, (and this without any reference to increased or decreased velocity of circulation,) but the *above* phenomena are attributable *solely* to the presence of *arterial blood*. Now, as arterial blood will become *depreciated* by a very slow circulation, or by the arrest of circulation, (as by confining the blood in a finger, by ligature, when the part included is seen to become *blue*, to *lose* its *sensibility* and *temperature*, and to afford *dark blood* upon being cut,) we may infer, that inflammation, which is characterized by the *opposite* phenomena of *increase* of *temperature*, *sensibility* and *colour*, should be owing to a *speedier circulation* of the vital fluid *through the part;* or, if the inflammatory action go on and become general, of a speedier circulation *throughout the system*, when the condition will be indicated by a quickened respiration and a more frequent pulse. This increased momentum of the blood would seem, *within* certain limits, to be in proportion to the intensity of the inflammation ; and if " the intensity of the inflammation carry it *beyond* a certain point, and the part come to *mortify*," I suppose this consequence to ensue, both here, and in all the other assigned causes for gangrene, because the circulation being *now* prevented, the *vitality* dependent on that circulation must also cease, or the death of the part be effected. But death and mortification are not the same thing, the now dead part must first have undergone the *action* which kills, or mortification.

Since, therefore, in order to produce gangrene, a suspended circulation is required, together probably with some other *peculiar* morbid action of the part, two questions come to present themselves, which have by no means met with that attention from pathologists that their importance demands.

1. *How* does intense inflammation, in the first place,

lead to the delay of the circulation? how is it, not a spur but a drag-chain? and,

2. By what immediate cause, or efficient, is the act of mortification set up?

In reply to the first question, it may probably be sufficient to observe for the present, that although inflammation *seems* to be *attended with* a quicker circulation at first, yet, if the exciting cause be violent, and the "appel des fluides" so great as to overcharge, and force into unnatural dilatation, rather perhaps, than stimulate a given structure, then the vessels will come to press upon each other, the flow of blood through them will be impeded, and the vital fluid, gradually accumulating, must shortly come almost to *stagnate* in such structure; discolouration, loss of sensibility, decrease of temperature follow, and the tendency towards mortification has commenced. In this way then it occasionally happens, that an organ under inflammation (*i. e.* an organ whose vitality was at first raised) may come to have no action at all, that is, congestion, the precursor of gangrene, will take place; such congestion being, as I conceive, as indispensable to the production of *all* gangrene, whatever be its more remote cause, as it is the *obvious* condition of a part, which, under tight compression, we may compel or precipitate into gangrene. The part into which the blood may arrive but cannot return, necessarily perishes.

Gangrene, however, we read, "has other efficients than inflammation;" that it has other *remote* and *predisposing* causes would be a statement more in accordance with *my* ideas. Thus, when gangrene is said to be brought on by chemical agents (where the vital chemistry of the blood is so altered and deteriorated, as to act no longer as a stimulus to the capillaries) the condition of the part as to *congestion* may be precisely the same, as where a very violent phlogosis has preceded, and the first condition, on my supposition, preparatory to gangrene, may be equally effected. Ossification of some of the arteries has been reckoned, for instance, as a cause of

gangrene. But ossification may be easily conceived to act on the principle of congestion previously formed in the part. In short, whatever has a tendency to produce stagnation has a tendency (that is, acts as a *predisposing* cause) towards the generation of gangrene.

The second question which we have yet to consider is, what may be the *immediate* cause, the real *efficient* of that action which terminates in gangrene? A portion of the blood sent from the common source of animal temperature and life, that is, from the heart and lungs, destined to carry vitality and heat over the body, is, under some of the foregoing circumstances, let us suppose, arrested in its return; it speedily imparts the vivifying principle with which it has been furnished to the texture in which it has, by supposition, become included; it loses this principle, ceases to be arterial, and becomes wholly unfit for the office of furnishing heat and maintaining sensibility. The animal temperature, the common measure of vitality, declines; *fresh* arterial blood is prevented from coming to the resuscitation of the part; while a *noxious influence* from the now *venalized blood* may possibly act upon it after the manner of an applied poison. The brain, we know, is incapable of sustaining the circulation of venous blood, as proved by asphyxia; the vehicle of *life* has become an agent of destruction, and a directly poisonous influence may concur with the interception of fresh supplies of healthy blood in the result that follows. The mortified part has not then passively ceased to exist, but has been submitted to an influence or an agency, which I have elsewhere termed *mortifactive*. Gangrene *may* be a sort of "*morbus venenatus*," originating in the noxious properties of undecarbonized blood, by *which* blood, the parts are stimulated to the last action of which their expiring energies are capable. Surely if the vitality of the *whole*, the *aggregate* of functional life, require arterial blood for *its* maintenance, it will not seem extravagant to hold, that the life of *each* organ derives its individual supplies from the same living current, and

that it will perish, when not only all further supplies are cut off, but when, moreover, the existing ones are poisoned.*

If, dismissing the pathology, we reason from the therapeutic of gangrene, there may be further grounds for considering the variety of supposed *causes*, as only remotely concerned in its production. The *treatment* of gangrene, as Mr. Laurence and others affirm, "must vary greatly according to the cause producing it, so that sometimes it will be found necessary to bleed, sometimes to give stimuli and tonics;" is this, I would ask, to make gangrene *one* and the *same*, or *two* distinct and *opposite* morbid actions? Can the *same action* require two modes of treatment so diametrically opposed as venesection and stimulation? this confusion, made so strikingly apparent when we come to discuss the remedial means to be employed, is to be found, I conceive, in the plain fact, that gangrene, when *conjoined* with inflammation, requires one line of treatment, and when it coexists with *another* state of parts, or of the system, another. Neither *bleeding*, nor *stimuli*, act *directly* upon the process of gangrene so as to arrest it *at once;* the *first* counteracts the violence of the inflammation (for which it is, in fact, a specific) and by doing

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* It is quite the opinion of modern physiologists, that unarterialized blood thrown upon the brain (the heart continuing to act awhile after respiration has ceased,) becomes the cause of death to the sensorium, and kills as a poison applied to the common origin of the nervous system. In the tragedy of Ion, Euripides embellishes an Epichorial fable which represents Minerva making a present to Ericthonius of two drops of blood—of the blood of Medusa, whom she had slain—τον μεν θανασιμον τον δε ακεσφορον νοσων, the one a poison the other a remedy—and it is curious that one of these drops was *venous* and the other *arterial.* Though the version of this story in Apollodorus unfortunately makes the *poisonous* drop to have issued from the *artery*, and the medicinal one from the *vein*, yet *any* fiction of this kind is singular enough, as it seems to intimate, that the ancients had a notion of a certain difference between the blood in the two systems of vessels. In like manner, the vest impregnated with the blood of Nessus, which Dejaneira presented to Hercules, seems to show, that they apprehended this fluid to be susceptible of highly morbid changes. The ancients knew nothing of *mineral* and very little of *vegetable* poisons. They sought for poisons in the *animal* kingdom, found them in the toad, and in hippomanes, and fancied them in *human blood.* Of poisonous snakes their knowledge was very limited, for these do not exist either in Greece or Italy.

so restores a healthy circulation and prevents congestion. Stimuli, moreover, should remove congestion when this is produced by *natural weakness* in the action of vessels. Thus remedies of an opposite class may tend to the same result, namely, to remove the *congestion* which precedes, and leads to destruction of the part. For gangrene, as gangrene, we have no remedy; but by knowing what *precedes*, we are enabled to oppose it upon philosophical principles; and, though the *act of mortifaction* be the same in all cases, may perceive why in some, bleeding, in others stimuli, must be resorted to. If the supposition of a diminished action of minute vessels in a state of enlargement, at first, on this account, more freely transmitting the blood, and so increasing the circulation in a part, but soon, from their augmented number and dimensions, and from the inflamed part being confined by contiguous parts, or compressed by the integuments, obstructing and impeding that circulation, make any approach to the true doctrine of inflammation, our practice, especially where gangrene is threatened, can neither be empirical nor dubious; for I entirely coincide with my friend Dr. Philips,* that in inflammation action is weakened, in gangrene destroyed. But I would go further; debility is not disease; death, or extinction of action, can only happen from disease. To me it seems too mechanical a view of gangrene, to suppose the death of a part to result from a mere physical impediment. At any rate, in a practical point of view, we shall scarcely err. Are parts debilitated and unable to forward their contents? To stimulate such parts were like attempting to resuscitate a drowned man by electricity. To excite an action which they cannot support, in *weakened* parts, is only to precipitate mischief. In such a case we unload vessels, and thus give them the only chance of recovering their tone, which if they do not, they must again be distended, and again emptied, till the powers of life becoming more and more inade-

* Essay on Inflammation, by P. L. Philips, M. D., 1833.

quate, the blood stagnates, and gangrene is the inevitable consequence.

In concluding these observations on gangrene, I may remark, that the circumstance of the circulation being nearly suspended in hybernating animals, and quite suspended in parts that have become frost-bitten; in the first of which cases it may be objected that mortification never happens, and in the second not of necessity, does not in reality make against my views concerning the deleterious influence of venous blood on parts in which it may be confined. For, in the first case, (hybernation,) the stoppage is never complete; the circulation is *uniformly* retarded over the *whole* body, so that no part can be said to be *congested;* add to which that there is in this instance a corresponding diminution of *animal and organic life,* in strict relation with the diminished *powers* of the bodily organs; in short, hybernation is not disease. And in the second case, (that of frost-bitten parts,) where congestion of vessels may be supposed, these parts possibly do not mortify, for the very reason that *they are frozen.* Their congealing may prevent their sloughing, by rendering them *incapable of action,* and the *nature* of their tendency to mortification may be illustrated by the facility with which they are observed to fall into that state on being precipitately thawed.*

ULCERATION.

After what has been already said of suppuration and gangrene, and the reasons which dispose me not to consider them as *mere* inflammatory products, I need say

* I may here remark, that Hunter's experiments on the blood, by which he attempted to establish its *vitality* or loss of life, from the comparative facility or difficulty with which it underwent congelation, by no means proves what this able experimenter intended. Since blood *may* be frozen *in the body,* (witness his own experiments on rabbits' ears, among other facts) and yet, when thawed again,

little respecting ulceration, to which the same objections will be found strictly applicable. "Ulceration," says Chomel, "must be admitted to depend upon conditions which we *cannot communicate* by artificial excitement, and which are neither to be looked for in the *intensity* or *duration* of an inflammation." If, then, so moderate a proselyte of Broussais, is compelled to admit that we *cannot* produce ulcers by first inducing inflammation, and if inflammation variously produced by nature, does not with any certainty conduct to this result, either by its *duration* or its intensity, such an admission seems sufficient to exclude inflammation from *any* connexion with ulcer. Nothing is more familiar, than that ulcers unpreceded by inflammation, whether symptoms during life, or visible evidence of its occupation after death, be appealed to, have their seat in almost every part of the body. This fact, I believe, is universally admitted by all who cannot consent at once to part with the convictions of sense and the deductions of reason, in deference to a vague hypothesis which would qualify inflammation in *such* cases with the *satisfactory* epithet of *latent*. Ulcer is, perhaps, seldom found even to coexist with proper inflammation; and, when this does happen to take place, it is well known that we can subdue *it*, and yet leave the ulcerative action untouched. Nor may we, in considering this subject, forget that inflammation, when it occurs in secreting vessels, suspends their function, and arrests any further formation of fluid. If, then, inflammation exert this effect on the healthy *absorbent* system, (by which the mechanism of ulcer is, we know, executed,) should we not presume, from analogy, that inflammation would rather *prohibit*, than promote, the action in which ulcer consists? In his very definition of inflammation, Mr. Lawrence includes

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manifest the properties of life, it follows that frozen blood, is not necessarily *dead* blood, but merely blood of which the organic life is for the time suspended. The power of resisting chemical and physical agencies is not the whole that enters into the idea of *vitality*, nor is a partial submission to them a proof that vitality is extinct.

the *impaired* or *suspended function* of the phlogosed part; the progressive absorption in which ulceration consists seems, therefore, so far from requiring, that it should be *suspended* by inflammation.

RAMOLLISSEMENT.

As to the *softening* of tissues, (another supposed result of inflammation, even from the days of Aretæus,) this change, so often noticed by the modern pathologist, may, I think, originate from *three* different sources, not one of which can be considered as inflammatory. As the hardening of a tissue seems to imply the conveyance of new matter into it, the opposite change must suppose the removal of something which gave it cohesion. Ramollissement, then, may proceed, 1st, from an *over-action* in the vessels that have that office to execute, *i.e.* from an augmented interstitial absorption. Or, 2d, it may arise from a diminished action of the vessels which *convey*, while those which remove, continue their function as before. Or, 3d, it may originate in some morbid condition of the material of nutrition in the blood itself. Andral's excellent judgment plainly detected the absurdity of consenting to ascribe some softenings of parts, to inflammation. "Who would venture to affirm," he asks, "that softenings of the brain under certain circumstances, or of the mucous membrane of the intestines, (*pale* withal, and *atrophied* in many diseases,) or the perforation of the cornea, (which, in the experiments of Magendie often happened when animals were exclusively confined to one species of diet,) or that softening of the heart or liver, accompanied with a *bloodless* condition of these viscera; or that the diminished cohesion which occurs in the osseous structure, constituting rickets, are common results of *one* cause, and that cause *inflammation?*" Some explanation of ramollisse-

ment in general, is indeed attempted by this able writer, who seldom permits himself to wander into the regions of hypothesis, by suggesting, that "when the two principal agents of life, the *blood* and the *nervous system*, have become unable, the one to *nourish*, the other to *excite*, the various textures of the body, a diminished cohesion between the component particles of organs, (brought to pass with more or less rapidity, according to the original compactness, or reverse, of the structure,) will at length conduct to the softening of the whole tissue.

Some writers, indeed, would infer *all* softening, to be always, because it is undoubtedly, they say, *sometimes*, inflammatory. "Il est rare," observes Condillac, "que les hommes ne se contentent pas d'une *première* reponse, et on diroit que leur curiosite les porte moins à s'instruire d'une chose, qu' à faire des questions sur plusieurs." Andral, however, does hold the opinion that softening of tissues may sometimes be even *proved* to be the result of preceding phlogosis; adducing as reasons, first, the anatomical examination of the parts so softened; second, the symptoms observed pending the deterioration of the structure; third, the causes concerned in producing it; and fourth, the kind of treatment most successfully opposed to its progress. But, I have taken occasion, more than once in this paper, to suggest, 1st, that *post mortem* appearances derive all their conclusiveness from the *symptoms* which precede or attend *them*, and that this *reflected* interpretation is lost as soon as it has become apparent that symptoms, in various cases of the same morbid change, have not been rigorously the same. 2d. The presumption of the inflammatory nature of ramollissement from symptoms, can proceed no further than to point to an occasional coexistence, which is not connexion—consequently, no sure conclusion on the nature of the process can be derived from inquiries of this kind. 3d. As to the argument derived from taking in the *causes* calculated to produce at once softening and inflammation,

it seems to be of very small value indeed. Does cold, which produces inflammation of the bowels, tend to produce *softening* of their mucous membrane? While those who say, that by inducing inflammation in the *first* instance, *this* will come in its turn to produce the softening, make an evident "*petitio principii.*" A corrosive substance administered to animals, doubtless gives rise to immediate symptoms of *inflammation;* and *ramollissement*, in such cases, after death is general. But is it necessary to admit, with Andral, that the first action in this case gave *origin* to the second, or is it not even certain that the corrosive substance which produces inflammation is here the *cause* of the softening? for if, when we apply the corrosive substance to the dead fibre, where inflammation is *impossible*, such softening ensues, it follows, that inflammation need not be the efficient of softening *when* the two effects concur. 4. The weakness of the argument from modes of treatment *said* to be found most beneficial, is obvious: antiphlogistic treatment may indeed be found advisable and efficient, *if* any inflammation attend ramollissement, but we have little proof of the influence of any treatment upon the *ramollissement* itself, since the only instances, where we can ascertain the existence of such a state at all, have been fatal cases. Tonics and stimulants introduced into a tender or irritated stomach, may indeed produce gastro-enterite, as the French writers say, but the obvious query here is, how do we know that in cases where we find symptoms of such a state succeed the internal application of stimuli, that a softening of the tissues of the alimentary canal *was* a previous condition of the parts?

INDURATION.

INDURATION can still less than ramollissement be considered as a termination of phlogosis, whether induration

of the bones (constituting the affection called by French writers éburnation) be intended, or of the muscular or nervous tissues, or of mucous or serous membranes, or of the parenchyma of any viscus, as of the heart, spleen, uterus, or *liver*.* The antiquity of this error constitutes its only claim to attention. There is no foundation for saying, that hardening, in *any* case, is produced or brought about by inflammation. Of course most of the arguments used above apply here; but, to take a case or two, if we find the *brain* hardened in confirmed *spirit drinkers*, no such inference can be drawn as that slow inflammation led to it, since we can produce the very same result on the *dead brain* by alcohol—leading to a suspicion at least, that, in such a case, the brain suffers a chemical, rather than a vital, action. But whether this be so or not, the cerebral mass is *often* found to harden as life advances, in consequence of some unknown action, which leaves inflammation at least out of the question. Thus bones get harder and more brittle as life goes on; but *not* from an increased circulation in vessels, since, on the authority of Munro, it is *difficult* to excite sufficient inflammation in these structures to furnish a supply of callus, with which, when broken, to unite their fractured extremities.

In a remarkable disease first noticed by Umbezius, a *general induration of the cellular membrane of infants*, produced, it would appear, by a superabundance of morbid secretion poured into its cells, no *inflammation* can be pretended; on the contrary, the infant becomes *cold* and *rigid*, livid in the face, and pulseless, and dies suffocated. Lastly, when *induration* occurs in the liver, spleen, pancreas or heart, "as we do not in the least

* The induration of the liver was distinctly ascribed to *phlogosis* by Aretæus, and, what is yet more remarkable, and in exact accordance with modern pathology, to phlogosis of a *moderated* or *chronic* kind. "If it be *violent*," he tells us, "it will conduct to *suppuration* of that viscus; but if its intensity diminish, what wonder that the organ should gradually become *hardened*? ην δε απο της φλεγμονης ανεκπυητον η το ηπαρ, απορον μεν ου γιγνεται, τον ογκον τι τον σκληρον ιζον εις σκιρρον ιδρυεται." He even mentions the sort of symptoms we may expect, which are those of *chronic inflammation*, "εστι πονος μεν ου ξυνεχης, νωθης δε.

know to what organic lesion of these organs inflammation really *does* conduct," (Chomel,) we cannot ascribe hardness to it.

HYPERTROPHY.

HYPERTROPHY may be of two kinds; first, it may consist in the increased specific gravity of a tissue, (which is so far an induration,) or in the thickening of a tissue from the secretion or addition of new materials; but, that neither case is of inflammatory origin is a point in which I agree with a good many writers, and disagree with Dr. Elliotson. The real cause of hypertrophy I believe to be simple hyperæmia,* by which *I* mean an excess, not necessarily of the *whole* blood, but of that part of the blood which *nourishes* the organ on which the increase of growth takes place. I conceive that the *action* conducting to this effect cannot exist without a corresponding increase of the *materials* of nutrition supplied by the blood, that is without a hyperæmia of some sort. "A more active appropriation of the nutritive particles contained in the *ordinary* supply of blood," (Townsend in the new Encycloped. of Med.) is *not*, I conceive the circumstance required; for this idea contemplates a *redundancy* of materials in health, presented to the nourishing vessels, whereas they have, more probably, but an adjusted sufficiency to accomplish the ends of accretion and growth, and no more.

Any connexion of hypertrophy with the opposite state *anæmia*, appears to me a physical impossibility, though the contrary opinion is advanced by Dr. Townsend, who draws that inference chiefly from the want of *redness* discernible in certain hypertrophied parts. This opinion

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* απο περιουσης αιματος εφ'η παχυνεται. Aret.

makes the state of *anæmia* to consist merely in the absence of the *colouring matter* of the blood; but as the botanist deduces no fact of *importance* from the *colour* of a plant, the pathologist should certainly not press this quality of parts too strongly in his arguments about their general condition—nimium ne credat *colori*. *Anæmia* is not, and should never be considered, as a deficiency of the red globules merely; it is, and ought to be regarded as a deficiency of that part, or of those constituents of the blood, and those only, on which vitality and growth are dependent. In *white* hypertrophy of the liver, *hyperæmia*, not *anæmia*, must be supposed, though a remarkable absence of *red* blood be conspicuous in the part which is the seat of the hypertrophy. Want of colour, then, not being a proof of *anæmia* in any legitimate sense of the term, *anæmia* can only mean a deficiency of *that* part of the blood on which growth or nutrition depend, without any respect to the colouring element. The latter may be deficient where the other may abound greatly. For instance, in induration of the white part of the brain, or of the medullary substance of nerves, there must be an increased supply of *matter*, as well as an increased elaborative action, to assimilate that matter. Thus, in hypertrophy of the bones, (whether *éburnation*—an increase of their calcareous matter; or *rachitis*—an increase of their animal constituents); in hypertrophy of the thyroid gland among the weakly inhabitants of the Alps; in the thickened tongue and tumid lips of similarly constituted individuals elsewhere; in the swelling of joints, or of the mesenteric glands in the scrophulously disposed, &c. &c., we recognise at once an increased supply of a natural material, assimilated by an *increased* action, and yet not an *inflammatory* one—though I by no means *limit* inflammatory action, for reasons to be afterwards stated, to vessels that convey red blood only.

THE BUFFY COAT.

Nor can the buffy coat of the blood be considered as produced by, though it oftenest coexist with an inflammatory habit. It must surely have some other *unknown* efficient, since it is found to occur in all habits of body. It cannot even be considered pathognomonic of that state of the body which is called plethora, and which probably precedes all inflammatory action, for buffy blood is sometimes found after excessive depletion *only*. It is sometimes found to obtain, too, where the system is under the effect of *mercury*—in neither case, of course, can inflammation be appealed to, without subverting every notion of the nature of inflammation, and all treatment founded upon it.

ALBUMINOUS URINE.

Nor is the albuminous deposite in the urine a result of inflammation. Dr. Elliotson justly observes, "that it is even very common for persons to be cured of dropsy by *bleeding*, although no *albumen* could be discovered in the urine; while, on the other hand, when there has been albumen in the urine, persons have not been cured of dropsy by bleeding." Mercury, which makes the blood buffy, also makes the urine *albuminous*, according to Chevallier.

ORGANIC CHANGES.

Authors generally suppose that organic changes—degenerations of parts in colour, texture and consistence—are effected by inflammation acting on a previous hyperæmia of the tissue: a conclusion to which I can

by no means bring myself to accede. The molecular changes, occurring in organic disease, are indeed not easily traced, but the cellular membrane is perhaps the invariable seat of all such change. Now, though it is usual to hear of "diseases converting organs into textures of quite a different kind from that of which they were formerly composed," an *immediate conversion*, or metamorphosis of matter, such as the vulgar contemplate in *petrifactions*, &c., is of course physically impossible. When a new product is said to be formed by a new action set up in the vessels of a part, it is quite clear that these propositions can only be understood in one philosophical sense; for, since two species of matter cannot occupy the *same* place at the same time, it becomes evident that *absorption* of the one must occur in the exact ratio of the deposition of the other. Organic change, therefore, is a duplex process, consisting in the *synergy* (the term is not mine) of deposition, with the absorption, or removal, of matters already deposed. That inflammation may indeed be *present* in the neighbourhood of organic changes progressing in different tissues of the body, cannot be denied; but such inflammation has no claim to be considered as *leading* to any change of *any* kind; I say of *any* kind, though all the principal changes may, perhaps, be reduced to three heads. The first may include such changes as consist in an alteration of the normal constituents of a part; as where cartilage becomes bone, or mucous membrane assumes the appearance of skin; the second, such as are compatible with the conservation of the original tissue, now merely modified in certain of its physical properties—shape, volume, or consistence; while under the third we may place the development of some *new* production either within the interstices of a given tissue, or amidst the cellular membrane which connects it, or upon its surface—which production may be solid, as tubercle; or fluid, as melanosis; more or less organized and identified with the whole; or, as in hydatids, in possession of a parasitical life of its own. *Inflamma-*

tion, however, is so far from producing such organic change, that any such production will frequently be found to have *advanced*, before inflammation comes on, and will then be suspended; and so return, alternately with inflammation for many times. Inflammation occurs not unfrequently at the end, often *only* at the end of these affections; but even the precedence *of* inflammation to organic change, (since it may occur at *any* period *during* organic change, and at *no* period of necessity,) cannot be considered as the *cause* of organic change in the present state of our knowledge. In some general remarks on this subject we find Andral making the incautious observation, that since such changes are not always preceded by *inflammatory symptoms*, we must either admit that they are "*tantôt* le résultât d'une inflammation, et *tántot n'en dependent pas;* ou reconnàitre qu'ils ne sont pas moins des phlegmasies, bien qu'ils *existent sans symptomes;*" now this inflammation *without symptoms*, is again a doctrine as old as Aretæus, who tells us, that from this deficiency of symptoms, suppuration of the spleen is hard to make out. But we can avoid this dilemma, by adopting a pathology which excludes inflammation from *any* participation in these morbid productions. I cannot by the way help admiring, that the very accurate author already so often referred to, should have been betrayed into an inconsistency with himself, in another part of his works, where, recurring to the subject of morbid changes, he again inquires, "concerning the immediate cause of the great phenomenon of *transformation*, whether it is to be considered as the consequence of inflammation, either manifest or latent," when his own answer had been conclusive—"Plus on médite sur les causes des transformations de tissus, plus on reste *convaincus* qu'on ne doit voir en elles que des resultats d'une *aberration* de la nutrition normale, *précédé* souvent, mais non *constamment* ni *necessairement* par un travail d'irritation"—neither *constant* nor *necessary*, since, he adds, "in a great majority of cases, the observation of symptoms, the study of étiology, the inspection of bodies, the laws of embryo-

geny, and also of those laws which preside over the nutrition of the body generally, conduct to the conclusion that every possible transformation may take place, *without* preceding irritation." Again, "create an irritation, and by artificial means determine a flow of blood upon some particular tissue; irritate the part as you will, in every way in which it is possible to excite it, and let this irritation be of any *intensity* or *duration;* yet, unless under certain circumstances *which you cannot command*, you will not effect any of the changes that are contemplated."

Although any remarks of mine on this part of the subject must necessarily be brief and general, (for were I to take up each morbid change in detail, I should have to transgress the limits which I impose upon myself in the present essay,) I cannot yet forbear an observation or two upon "*tubercle*," concerning the formation of which we hear so many opinions advanced with equal confidence, and, as I think, for the most part with equal fallacy. I again quote the following passages from Andral:—"If the *disposition* to form *any* new product be very decided, in that case even the slightest congestion will be found sufficient for its manifestation. This is to be observed in what is called the *tubercular diathesis*. But if the *disposition* in question be less marked, it will then require a considerable congestion, and one of sufficient *permanence* to excite a degree of *inflammation* in the part. And lastly, if the *disposition* does not exist at all, then, even the most intense phlegmasia, and the most enduring, will not lead to tubercle." Inflammation then in his opinion, (very unlike to that of the Broussaists,) is powerless to produce tubercle, or indeed any other product, unless a constitutional *disposition* to its formation be present. For my part, and making small account of *any* condition of vessels, I should say that if there be no *material* of the kind in the blood, tubercle cannot be formed—on the principle that "ex nihilo nihil fit." But Andral, we find, thought fit to modify this opinion and give up congestion alto-

gether: "Je ne pense *plus aujourdhui* qui l'existence antécedente de cette hypéremie soit necessaire à la formation du tubercule; il y a *seulement perversion* de sa force normale de secretion; cette perversion elle même, peut être une produit *independante* d'une irritation antécedente." "Therefore," he concludes, "if we could *show*, that which we may at least suppose without absurdity, that the matter constituting tubercle is *formed in blood* become diseased, (as even in a state of health, the proximate principle of urine exists in the blood,) we may easily imagine a *deposition* of this matter into the structure of certain organs, in a manner purely mechanical, and without the aid even of previous *congestion*." A deposition of tubercular, I might add of all secreted matters, might evidently be brought about, either by some peculiarity in the action of vessels, such as to let them transmit parts of the blood as through a filter, and so change its *crasis;* or from a disposition in the blood itself in its passage through different organs, to give out, at one time its colouring matter, at another its fibrine, albumen, or salts; or, if diseased, perhaps new products, as pus, or the matter of tubercle. Even though tubercle, agreeably to another writer, consisted in the effusion of a coagulable lymph *deficient in vitality*, produced by a peculiar depravation of the function of assimilation, still we see nothing like inflammation in its production.

But to return to the *general* view of the subject, from which these remarks on the formation of tubercle have led me, I would ask, whether our very want of successful means with which to oppose them, together with the constant *obscurity* in the diagnosis of organic changes, does not go distinctly to prove them *not* inflammatory? Bleeding subdues inflammation, and takes off the load of blood in hyperæmia; but bleeding has little, if any, effect in stopping morbid growths. It can diminish the increased circulation arising from enlarged vessels distended by blood; (a condition which I shall presently attempt to

prove takes place in phlogosis;) it can check an action exaggerated in *degree*, but it can have no direct influence over an action unnatural in *mode*. Thus bleeding is a *specific* for inflammation, unmodified by syphilis; but in *syphilitic* Iritis in order to cure the compound affection, we require these two agents conjoined, the bleeding putting a stop to the inflammatory symptoms, while the syphilis in the blood is eradicated by the mercurial action. *All* diseases are probably *complicated*, and on their greater or less complication, and the proportion borne by the one condition which, perhaps, we may know how to treat, to some other with which it may be conjoined, and for which we may or may not have remedies, will our power of controlling, or curing malady in general be found to depend.

If I have shown that no disorganization can be fairly considered as a *product of inflammation*, whether acute or chronic; there being indeed, as was observed, no difference between the two but in amount, it is plain that the origin of all organic degeneration must be sought for elsewhere.

The common *source* of all organic changes, I conceive to reside in certain deleterious agents existing in the blood itself. Hyperæmia and inflammation may frequently *precede*, but *neither* can be charged with the *production* of organic change, since such change, as it is well known, requires neither intensity nor duration in a previous congestion, which, if it happen to exist, may be strong or feeble, long or short in its duration, and yet be followed indifferently by *every* change or by none. Certain *deleterious properties* then, and not *mere congestion* of the blood, whether in hyperæmia or inflammation, can scarcely fail to be concerned where organic mischiefs are in progress, and surely we have abundant certainty that the vital fluid, though to our *senses* healthy, may be charged with the elements of disease. Such degeneration of the blood may, perhaps, be conceived to occur in one of the three following ways :—
1. By the addition of certain new and unknown agents.

2. By the abduction of elements necessary to health. 3. By these two circumstances conjoined, as perhaps in epidemic cholera. An *increased supply*, however, of any *normal* constituent, which merely augments natural action, or that debility which appears to consist in a diminished supply of its appropriate stimulus to a part, are perhaps neither to be considered as states of actual disease, nor as leading, except very remotely, to that result. That the blood may *appear* healthy, and yet be diseased, does not admit of doubt, although our senses be unequal to the task of distinguishing, in most cases, any difference of appearance, and the aid of chemistry be equally unavailing.

Objections to an humoral pathology, properly *defined*, (and who that reflects much upon the subject can refuse to become to a certain extent an humoral pathologist?) derived from chemistry not revealing any depravation of the humours, should cease, when we reflect on the one hand, on the insufficiency of the most delicate tests to detect those miasmata and other subtle poisons which, entering into the blood, evade the most careful and elaborate analysis, and on the other, that no *analytical* difference is found, in bodies the most dissimilar to our senses, as for example, *starch* and *sugar*. "En vain le chymiste se flatte d'arriver par *l'analyse* aux premiers éléments : rien ne lui prouve que ce qu'il prend pour un élément simple et homogène ne soit pas un corps composé de principes hétérogenes, mais que la seule imperfection des instruments ne lui permet pas de décomposer davantage," is a remark that will *still* continue to apply to *animal* chemistry, even should the triumphs of the science over every other subject of its research be complete.* The objections, then, from chemistry

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* Dr. Stevens makes the *complexion* of the blood in cholera to depend entirely upon the absence of certain *saline* ingredients, and institutes experiments to prove it. May not this dark blood, however, be in a great measure, if not wholly, brought about by the necessary *approximation of the red globules*, when the *serum in which they are suspended is withdrawn?* The solid pigment of the clot is, and ought to be, darker than the same pigment *diluted in a nearly transparent medium.*

to a rightly and rationally entertained humoral pathology are of small weight, provided we be able to give any other grounds for adopting this much abused, yet certainly not unphilosophical doctrine. I shall limit myself here strictly to some few reflections of my own on this subject—a circumstance which I am bound to state, because, as my notions have been formed and matured without any extraneous assistance, it cannot be, but that I should have failed to acquire very many powerful arguments which must have occurred to others, and would come in aid of my own views; my object, however, is not to give a history of opinions, but rather, in as few words as possible, to submit my own thoughts to candid consideration.

It is scarcely, I think, to be doubted, that the healthy actions of the body, (which depend upon the solids for their performance,) are brought about by certain *vivifying principles in the blood*, which every-where pervades the finest structures, and through which the organs may be said to "live and *move* and have their being." It seems, moreover, highly probable, since the actions performed are so different, and since different effects imply different causes, that *different properties* of the blood are in demand, in different cases; that while one property may act as the appropriate stimulus of one organization, another property may awaken the energies of a second; and that there may exist as many individual and appropriate stimuli in the composition of healthy blood, as there are kinds of actions to be performed by a healthy body. In disease, then, when the actions are changed, either the infusion of some *new* element must be supposed, or the abduction of some old one, because quite *new* actions are often set up in the system, or the performance of usual ones *arrested;* and because *all* action implying stimulus, and the *natural actions* arising out of certain stimuli in the *blood*, various probably as themselves are various, it would be gratuitous to suppose that *morbid action* (*i. e.* disease) can have any other origin. Meckel, indeed, enlarging on a proposition

of Andral, maintains, that *all accidental formations*, whether of the same kind with any of the structures of the body, or new and foreign to it, are effected by the secretion of a peculiar fluid from the blood. I would go a step even beyond this; being disposed to believe, that after making all due allowance for the share which secerning vessels may take in the process of morbid changes, the blood itself, the living *organized* blood, may have a *greater* share in effecting a given change than that of merely affording the pabulum upon which, as some say, such change depends. It might do this, that is, it might furnish a material, if it were *not* a *vital* fluid; but since it has life, *precisely* in the same sense that the solids have life, what is more likely than that the results contemplated, should arise from synergic *actions* of the solids of the body, and of that blood, *by* which those solids are sustained, *out of* which they were moulded, and into which they are again to pass, before they are thrown off from the body? Since writing the above, I have been gratified in finding that conclusions have been elsewhere drawn, which imply at least a similar train of thought, and impart some additional plausibility to this speculation. If the reader will refer to the article *Urine*, furnished by Andral, in the Diction. de Medicine, he will find the following passage:—"Tous ces faits prouvent, et le resultat est déjà fort important, que la formation des divers principes immédiats qui composent nos liquides ne dépend pas tellement de la *structure d'un seul solide*, qu'on ne les voie se *sèparer* aussi, soit *normalement*, soit *accidentellement*, *d'autres solides de structure toute differente*." Yet how much rather may we attribute to the *blood* itself, than to the *vessels* which transmit it, the respective powers of forming, or imbibing, and that of separating and deposing a morbid principle under certain conditions? But whether we shall admit the existence of morbid elements in the blood, and hold them to be separable from it by an *intrinsic* action of its own, or whether we refuse this action to the vital fluid, and confer it on the *solids* (that is, on the

vessels) still, it is clear, that in the case of every *new product* we must infer a new, extraordinary, and *specific stimulation* to have preceded—that is, some *alteration* in the *constitution* of the blood. *Hyperæmia* may, by accumulating any new principle upon certain parts favourably circumstanced, promote or accelerate, but it cannot *produce* any morbid change to which a part may be submitted. *Hyperæmia* is not necessary to the conversion of cartilage into bone; in order to accomplish that change, the actual presence of a *new principle* is required; that principle must be *in* the blood, because the *same* vessels that formerly poured out the matter of cartilage now pour out the matter of bone; when results are thus altered, since there is no difference in the *vessels*, there must be a difference in their action; and how should that difference in action take place, but from the presence of a *new* or newly active principle or element in the blood? on the same principle, in morbid growths, it is not mere blood that is required, but blood of a particular quality; from such blood only can morbid deposition take place,—and this where original structure predisposes, or casual irritation invites.

But were *hyperæmia* admitted, as is maintained by some, to be absolutely indispensable to morbid accretions, we need not on that account abandon the opinion that such accretions are mainly attributable to changes in the *composition* of the blood, which hyperæmia, by concentrating on a part, may have rendered more effective. Hyperæmia itself is but an *effect*. In inquiring into the real efficient, the *point de depart*, of the redundant circulation or over nourishment of a part, may the speculation be hazarded of an inherent *irritability*, so to speak, of the *blood itself*, analogous to the *organic life* possessed by the solids, and susceptible, like it, of local accumulation, added to a loss of irritability in certain vessels, which thus become unable to oppose its ingress?

It is clear that in all local hyperæmia, that is, *wherever* there is real accumulation of blood in any part of the body, it must be either passively *drawn* there, or

urged by the *vis a tergo*, or *carry itself* there, so to speak, by its own act. In these latter times, when every thing is ascribed to the vitality of the solids, and the blood, out of which these solids are made, and by which they are supported, obtains the *otiose* dignity, (for nothing is meant by the concession,) of a *vital* fluid, it may be thought sufficient to say, that the vital energies of the solids are *exalted*, and *consequently* that blood, in the given circumstances, accumulates in them, either in the state required by inflammation, or in that of active hyperæmia. But how come the solids to *be* thus *exalted* in their vital energies, except by means of the vital fluid which feeds and stimulates them? Further, if the *mode* of action of blood-vessels was *expansive*, not *contractile*, there would be less difficulty in accepting increased vitality and activity of vessels, as the cause of an undue supply of blood, since this fluid would, on this concession, be invited into canals of *enlarged diameters*—an intelligible *appel des fluides*—or be pressed into them by the atmosphere. But since the action of vessels is precisely the opposite, any *increase* of their *vis propria* should be attended with a corresponding *increase of* the amount of *their contraction*, that is with a corresponding *diminution* of their capacity, which must needs tend, by the communication of *augmented velocity*, to accelerate the progress of the blood *out* of the part, in place of permitting the reception of augmented supplies *within* their canals. If, for instance, inflammation consisted in *over-action* of the vessels, and not on the *vis a tergo* acting on vessels of *enlarged diameters* and *deficient* energy, ought not inflamed parts to be characterized by diminished volume, an ex-sanguine state, and consequently by diminished sensibility and temperature; by conditions, in short, the very opposite of those which we refer to, as decisive of its presence? In short, in closely considering the kindred subjects of inflammation and hyperæmia, is it *intuitively* out of question, whether blood may not be accumulated in organs, in virtue of some attribute of its own, acting in concurrence, how-

ever, with the necessary conditions, *increased size*, and *diminished* contractile *energy* of vessels?

But, it is not because I might be disposed to suggest in the blood itself, at least, an assisting *cause* of hyperæmia, that I would look upon this fluid as affording the starting point of organic change, or of disease generally; I am far more strongly led to this sentiment by the consideration, that all the tissues of our body are formed out of blood, which probably contains the constituents of each, so beautifully and wonderfully blended together as to be quite insusceptible of any other chemistry than the chemistry of life. The blood exists in so great an excess over the mere solids of the body, that the *chances* of disease *originating* in it, should be also, we might conjecture, great in proportion; and these chances surely become much more numerous, when we consider that the blood is not only exposed to all the influences that *can* possibly affect the solids, but that it also constitutes the current by which effete matter is conveyed away from the body, as well as that by which new matters are received into it. Moreover, as blood cannot impart, as we think it does impart, their *vitality* to the solids without possessing it, the *life* of the blood, notwithstanding our habit of imputing life *only* to solid and contractile matter, seems to be a doctrine that even in *logic* cannot be impugned. "Beaucoup de liquides animales," says Breschat, and I am fully disposed to agree with him, "sont, suivant moi, *organisès*, et ne different des solides *que par leur densité*." To me, I own, that it seems far more probable, that this vital fluid, during the act of its formation out of so many heterogeneous materials, should imbibe certain deleterious matters foreign to its composition, which it may separate from itself during the circulation, and cast off, by an action mostly, if not altogether, its own, than that being incorruptible and unalterable in its *crasis*, the solids should *conjure* out of this blood, by a sort of magic, materials for the production of those countless changes which may obtain in the different tissues of the body.

Besides, in the *interior* of the body where the solids are out of the reach of all external agents, is it not almost of necessity that the first morbid impression, or *point de depart*, of disease should be conveyed to them through the circulating fluid?

Whatever theory we may adopt respecting the morbid changes which occur in different parts of the body, it is clear that even supposing inflammation, congestion, or hyperæmia, always to *precede* organic change, the mere accumulation of blood in a part, whatever be the cause determining it thither, cannot give rise to a change, for the existence of which we must, at least in relalation to some *species*, absolutely suppose a new element, in the blood. It is no argument indeed, but the treatment, if it may be called such, of organic disease, shows that a diseased condition of the blood has been always apprehended; for not to dwell on such terms as purifiers, sweeteners, &c., what do we mean when we talk so advisedly on the use of alteratives? what are alteratives? medicines that alter morbid into healthy action; but there should seem to be no way to alter morbid or abnormal action, but by altering the perverting stimulus which led to that action; and is not this stimulus in the blood? it is scarcely necessary again to revert to the supposed origin of morbid growths in hyperæmia pushed on to inflammation, though it cannot be overlooked, that mere bleeding will never remove them or check their progress, as it ought to do, if they were of the nature of inflammatory products. It is not denied that inflammation *may* attend the disposition to form morbid growths, but by gratuitously assuming, that in all cases it *must* attend, we should weaken the patient, by instituting the practice of depletion, without even approaching the real disease. Can, there, in fact, be greater absurdity than the desire of tracing all diseases to any one source? and is the universal agent employed by Broussais for this end, *call* it Inflammation, or Irritation, or *conceal* it under any other specious term—more logical, more easy to be believed, or more ex-

planatory, than the putridity of the earlier schools, or the still more ancient Cachexia, of which Aretæus speaks in the same inconclusive style?* Our submission to Broussaism would effectually prevent our even seeking in the exhaustless stores of nature for those *specific* remedies, which, there is every reason to hope, will, either by diligence, experience, or accident, be in time multiplied in our hands; for if all diseases can be traced up into one action, all remedies might be comprised in one class—those which coerce inflammation.

It is indeed true, that while we administer with becoming confidence a few active medicines of general powers, and with becoming scepticism such as enjoy a reputation for specific ones, we are ever on the look out for the aggravation of disease by *fever* or *inflammation*, and are not sorry to find occasion to bleed, because active disease is more welcome than passive, and because bleeding is, beyond all question, the most powerful of all our means of changing the actions of parts. As to alteratives, (the *expletives* of medical routine,) if this term attest the possession of any quality at all, is it not that of influencing *the blood*, either by communicating to it a new principle, or by changing and making inert the morbid principle on which the development and progress of diseased action depend,—as some have attributed the power of mercury in acute inflammatory disease, (croup for instance,) to its checking the disposition to throw out coagulable lymph.

Finally, and to conclude this subject of morbid growths, I would further argue against the Broussiasts, and I think conclusively, that if inflammation be only a *natural* action of a particular set of vessels, as I believe it to be; and the action conducting to morbid growth *another* action; as two actions cannot go on in the same *vessels* at the same time, it must follow, either that morbid growths, if effected by the mechanism of vessels at all, are not effected by their capillary extremities, the seat of

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"Ξυμπαντων ομου παθεων καχ ιξιη τροπη. απασαι γαρ αι νουσοι, τησδε αποτοκοι."

inflammation; or else that this action *terminates*, before, by a *new* action, they are in a condition to eliminate that morbid principle of the blood which accident shall have introduced into it.

I shall now say a few words on INFLAMMATION itself.

(1.) I began this paper by stating my opinion, that inflammation is always the *same* action on whatever parts or tissues it may make its appearance, and that the different phenomena which are wont to attend its beginning, progress, or termination, depend on some unexplained agency. Not only all the diseases technically called phlegmasiæ, but all disease whatever, may be *complicated* with inflammation. But 1st, as inflammation, it has been shown, may exist without its alleged products, and these products without inflammation, inflammation is not properly their efficient; 2d, the great *dissimilitude* of so called inflammatory results, proving that they have an *equal* right, proves too much, and divests them of any right to be traced to *one* agent; and 3d, the total want of *proportion* between the amount of inflammation and that of its reputed results, conducts to the same conclusion. The *variety* of treatment moreover required for many of the phlegmasiæ, (for instance those of the serous, and those of the mucous membranes; of the fleshy viscera, or of the skin;) the impossibility of arresting many of them by any vigour of *antiphlogistic* treatment; and finally the frequent absence of *constitutional symptoms* during their progress, tend to the same conclusion and point out pretty distinctly the looseness of our pathology. That the treatment of inflammation may be very *different* in different cases, and yet equally successful, by no means proves the affection, *inflammation*, to be different, except in *degree;* for, admitting that in some cases we administer tonics and astringents with advantage, while others imperatively demand bleeding and other depletive remedies, in both cases we act, or should act with the *same* intention—only taking different modes to arrive at the same result, and our professional skill being put to the test, by the judgment we

exercise in the selection of the right mode, *under the circumstances*. To relieve distended blood-vessels, and restore their diameter to their *natural standard*, is, in inflammation, the one and universal indication. If their vital energy, their excitability, require a *spur*, we have remedies or applications, by the aid of which, the blood which has forcibly distended the capillaries, is urged forward; having been thus enabled to relieve themselves, these vessels will now perhaps offer more resistance to the blood still sent upon them from behind; on the other hand, if the capillaries be already too much debilitated by distention too long unrelieved, stimuli will certainly prove injurious, since to excite unproductive action in weakened parts, is futile, or mischievous. Yet even here, depletion may still have a place, and by a very cautious and moderate application of leeches, we may afford to parts in such unpromising condition, the only chance of recovering their *tone*, which, if they now fail to do, the hazard of sloughing or sphacelation is at hand.

(2.) Inflammation appears to me to have its "*point de depart*," its starting point, always in the blood itself, nor does the circumstance of its occupation of a limited space by any means disturb this conclusion; for, is it more easy to account for *local* inflammation, by conceiving it to originate in a local act of the *nerves*, or of the *nervous influence*, upon blood-vessels? If the blood, as it may be said, is every-where, the ubiquity of *vessels*, is just as certain. If it be again objected that the blood is continually *changing its place*, and therefore, if any influence inherent in it could produce inflammation, this influence should act every-where, it may be replied that the ubiquity of the blood need not suppose a general spread of the diseased action it may still give origin to; blood, which has conceived or obtained the principle of inflammation, has necessarily passed through some vessels before it comes into contact with others; those against which it happens *first* to impinge, may deprive it of its *acrimonious* or *exciting* character, and the

immunity of the whole vascular system be purchased at the expense of a part. But, it may again be objected, if the incentive to inflammation really were in the blood, it should be found to *follow its course*, and yet in phlebitis, as Mr. Breschet observes, "l'inflammation ne se develope pas toujours en suivant le cours du sang dans les vaisseaux. J'ai vu plusieurs fois cette phlegmasie aller dans une direction *opposée* à celle de la circulation sanguine dans les veines." Here, however, the writer would appear to overlook, that in *phlebitis* it is the "*vasa vasorum*" which inflame, and that consequently, from the variety of anastomoses maintained between these minute vessels, the inflammation *may spread against the general current of the blood*, and *yet* not against the direction of that portion of it which circulates in their coats, and which alone may be concerned. If inflammation did not begin in the blood, but were set up by any act originating in the nervous system, might we not reasonably expect, that under certain circumstances, some casual actions or affections of the nerves would control or modify it? thus the process of secretion, which is to a certain extent at least, under nervous influence, becomes disturbed in violent mental emotions—but who ever heard of inflammation being made less intense by *fear*, or aggravated by *anger?*—not to say that the occasional absence, or insignificance of pain, even in acute inflammation, seems to throw its production rather upon some reciprocal action of the blood and its vessels, than upon any undue activity or influence of the nerves.

(3.) Under inflammation, the blood accumulates in a part either by a *vis a tergo*, or as seems more probable, by a diminished vitality in the vessels of the inflamed structure. Impulse from behind, as elsewhere observed, is a more probable agent than attraction to a point through nervous excitement; because, the probabilities of either being supposed equal a priori, any superinduced irritability of the minuter vessels would rather tend to *extrude* the blood already in their canals, than dispose

them to imbibe more. If a vitality in the blood, embracing *all* the conditions of life in the solids, were supposable, then this fluid might indeed flow to a part by an act of its own—but this is not a doctrine that the most decided theorist could yet venture to propose.

(4.) In inflammation, within certain limits, the *action* of the inflamed vessels is diminished, although the *circulation* is accelerated; for increased pulsation, under inflammation, is no proof of increased *action* of the arteries, as it may merely result from augmented *size* of these vessels. The *over-action* of an artery is, besides, one thing, and the *acceleration* of the circulation through that artery another, though the last, as I believe, actually exists, at the commencement at least, of all inflammation, because of the increased diameter which the very minute vessels acquire: for, though Dr. Young proved the *friction* to be inconsiderable in the order of vessels which he examined, yet it may be very *great* in vessels still *smaller* than these, and in fact can scarce be otherwise, when the approximation of the fluid globules to the solid material within which they circulate, brings them within the sphere of mutual attraction. Thus, I conceive, that an augmented momentum may be acquired by the blood in an inflamed part, in the simple fact of the dilatation of very minute vessels, such expansion rendering their capillary bores equal in size to the arteries from which they proceed, and the veins into which they open.

(5.) Beyond certain limits of endurance or resistance, which vary in different constitutions and habits of body, the action of minuter vessels under inflammation becomes more and more weakened, and if the sense of distention be very great, is at length quite destroyed, the derangement proceeding from a *diminished* to a *suspended* circulation, and ultimately to gangrene. Such a view of inflammation would at least reconcile the apparent discrepancy of authors, some of whom affirm that it quickens, others, that it retards the circulation. It may do either; in fact the same inflammation frequently does both,

and in an inconceivably short space of time. When I speak of a diminished action of minute vessels, as occurring in inflammation, of course I use the words in a relative not in a positive sense. Phlogosis, when it takes place in *weakly and cachectic habits of body*, is more prone to run into gangrene than when the constitution is unimpaired, perhaps because the healthy action of the part being originally feeble, it becomes less difficult for disease to suspend it altogether, or to substitute a new and a morbid action in its stead, than if the normal action of the part had been more vigorous. We might perhaps cite, in further support of the opinion which regards inflammation as attended with diminished action of vessels, that inflamed glands do not secrete ; a better reason, however, for a suspended secretion is, I think, to be found in the *introduction of the wrong material* for forming the peculiar fluid of each gland ; or in the material, under the circumstances, not being conveniently, or properly presented. I conceive that blood, *unprepared* and *undivided*, may possibly, under inflammation, be forced into secerning vessels, which in the healthy state are fitted to act on *certain elements* only, and conclude that if the right elements are not presented to the vessels, they necessarily suspend their function. Mr. Laurence has indeed truly observed, that we need not have recourse to a supposed *spasm* of the capillary vessels of glands, to explain the phenomena of arrested secretion, but, since to say that "they are disturbed in their action," or that they are "thrown into an unnatural state," is but the expression of the fact, and since, as physiologists, we should inquire *how* they are disturbed, and *why* they are in this unnatural state, the supposition of an unprepared material being forced upon them, may, perhaps, be not irrationally entertained.

(6.) Inflammation is, I conceive, a *natural* action, diminished in degree ; but not, unless I reason inconclusively, altered, as Mr. Laurence states, *in mode*. This may indeed, as I think, be proved, by what the French writers call the "voie d'exclusion," almost, indeed, by the

"reductio ad absurdum" of mathematicians, and if the whole of this paper has not led indirectly to this conclusion, other circumstances are still at hand to supply the same inference. 1. If inflammation were "increased vital action altered *in mode*," we should probably not be able to effect that very speedy *relief* which we do effect by bleeding; for bleeding merely *diminishes* the momentum of the circulating fluid, by unloading the vessels and so reducing their diameters, but can surely not exercise any immediate control over an *action* altered in *mode*. 2. Dr. Elliotson has very truly observed, that we often fail to find, after unequivocal symptoms of internal inflammation, any vestiges of it on dissection. Now with a natural action disturbed merely in *degree*, this nullity of result seems less surprising, while of an action altered in mode, the consequences could not be expected to disappear after death. 3. That inflammation is a *natural* action, not altered *in mode* is further probable from its *sudden metastasis* from one part of the body to another. 4. A similar presumption is afforded by its *sudden disappearance* from a part where it has existed for some time. And 5, from its revisiting certain parts *periodically*—a fact, I believe, rigorously ascertained. In *all* these instances the parts which the inflammation has abandoned, are admitted to be found in a perfectly healthy and normal condition.

(7.) Inflammation, I further conceive, commences always in the red *capillaries*, and is *restricted*, at its commencement, to these vessels exclusively. This statement, I am aware, is at direct variance with all French pathology, which expressly advocates, and speaks familiarly of an "inflammation des vaisseaux *blancs*." But, as the only known symptoms of inflammation, are pain, *redness*, *heat*, and *swelling*, how, it may be fairly inquired, can *redness* exist without the presence of those globules which are the vehicle of that colour, or how can augmented temperature (which, from the beautiful experiments of Edwards and others, appears to bear a direct ratio to the *amount of these globules*) take place, when they are excluded? the

two other symptoms, pain and swelling, are neither by any means constant, nor if ever so constant, could they be received alone as adequate proofs even of the *existence*, far less of the *nature* of a travail inflammatoire.

(8.) But inflammation begun in the *red capillaries* may be carried beyond its natural limits, and the augmented momentum with which the blood is moved onwards may at length come to inject vessels, which, in their natural state, transmit only a *colourless* fluid through their canals. It cannot, however, *begin* in these transparent vessels—unless *redness* and *heat* be also excluded from the symptoms of inflammation.

(9.) Inflammation cannot, in strict language, *begin* in *serous* membranes, nor yet be *propagated* to these membranes. The evidence on this subject, I think, is complete, and deducible, both from anatomical considerations, *post mortem* phenomena, and sometimes even from the absence of symptoms. The *anatomical* evidence could not well be stronger. Bichat, the most accurate of anatomists, has said, "that all *serous* membranes indifferently, are supplied with two orders of vessels, which are wholly *their own*, and isolated, as it were, from the blood-vessels of the system; these vessels are lymphatics and *absorbents;* neither of them *continuous* with *red arteries*, nor, indeed, with any other order of vessels." *If* this statement be correct, which, however, I am aware that some deny, it follows irresistibly that they cannot be injected with *red blood*, and consequently that they cannot *inflame*. But the question seems placed beyond dispute by the inspection of the bodies of those who have died of what *is called* an inflammation of the serous membrane, whether it be of the pleura, the pericardium, or the peritoneum. I shall, on this point, content myself with the single but sufficient testimony of Chomel, though Andral and others might be cited to the same effect. "Les membranes sereuses n'offrent *jamais* de changemens appréciables dans leur èpaisseur et dans leur texture; elles paraissent *emprunter au tissue qui les unit aux* parties voisines, la *nuance rougeatre* qu'elles offrent *quelquefois*, et qui

cesse presque entierement d'etre sensible quand on les examine *au jour*, après les avoir séparées de ce tissu. Leur épaississement est si rare, que je n'en ai pas rencontré *un seul example.*" He adds, "C'est dont uniquement d'àprés les changemeiis survenus dans le produit de leur *exhalation*, qu'on peut reconnaitre, en anatomie pathologique, l'inflammation des membranes sereuses." But we have, I trust already seen good reason for *not* assuming any changes in exhalation, in proof of an *inflammatory* action, for the existence of which we cannot offer other and better evidence. Nor can the formation of false membranes, by which opposite surfaces occasionally become connected, (even should we admit the existence of such membranes in general to imply inflammation,) be assumed in proof, that any *serous* membrane has been *substantially* phlogosed; because the effusion of a fluid, liable to be subsequently consolidated, may be effected through openings in the serous membranes, like *pores* in the skin, just as well as by the ramification of vessels through their substance. In correct language, therefore, I apprehend that persons cannot be said to die of inflamed pericardium, inflamed pleura, or inflamed peritoneum; but whether, as Pinel thought, there exist in such cases, really *two* diseases, an original and constitutional one, to which certain local symptoms are but secondary and unessential; or that a local affection, in the first instance, is productive of a fatal constitutional disturbance, seems very immaterial in fact, and is quite so to my argument. All that I contend for is, that the seat of inflammation in these diseases, may be *below* and about, but is not properly *in*, or of, the *serous* membranes. Even pain referred to their situation, may arise from the tension of *subjacent* nervous filaments, under the pressure of turgescent blood-vessels, with which they are in immediate proximity or contact, and in which the 'travail inflammatoire' may exclusively exist. Since meditating the above sentences I have been gratified to find that I am far from being alone, in denying the capacity of pure

inflammation to *serous* membranes. Olivier, a pathologist of high authority, announces a very distinct opinion on this head, and states that other authors have seen reasons for the same conclusion, although these reasons, either for their, or his own opinion, are not stated, as they surely ought to have been, together with the following unequivocal declaration of it:—"L'inflammation des membranes séreuses n'a point, a proprement parler, son siège dans *ces membranes elles-mêmes*, mais dans le tissu cellulaire qui leur est sous-jacent, ou dans les couches les plus superficelles des tissus qu'elles tapissent. L'homogenêité de leur texture, *qui offre a peine des traces d'organization*, appuie cet opinion, qui a été soutenue long temps par Rudolphe, Chaussier, et Ribes, et que je me suis aussi attaché a demontrer, en traitant de la meningite rachidienne, dans mon traité de la Moelle épinière et de ses maladies." I regret the being unable to consult this well known work at the present moment.

I cannot, however, admit with this writer, that the *white* vessels which ramify through the substance of serous membranes, can be distended by red blood, "*quand* l'inflammation *y fait penetrer* le sang." *If* this ever occur, since there is no *continuity* of vascular communication between these membranes and those parts with which they are in contact, it must rather be by *effusion* and consequent *absorption*, or by a species of *endosmose* as it is called, *through* the membrane. Nor is the opinion I have ventured to express, that the so called inflammation of serous membrane, is confined to parts contiguous to it, and never, in reality, pervades the *substance* of the membrane, without support in the admitted fact, that no blood has ever been detected in the substance of the *arachnoid* either in health or disease. The arachnoid surely would *exhibit* inflammation, if inflammation could exist in it, and if it do not even *seem* to inflame, while other serous membranes do, it is because they are more *intimately connected* than it, with very *vascular* parts. Applying of course, to the *synovial* serous membranes, the observations just made of the *splanchnic*, the

painful affections of which *these* may become the seats, either where they cover articulations, or tendons, or elsewhere, are not, as I conceive, strictly of the *nature* of inflammation—for *pain* does not imply inflammation. Nor, again, do I see how dropsy of a serous membrane could have any right ever to be considered as an inflammatory affection, even if these textures were supposed capable of inflammation, for in such dropsies the membranes do not even appear *reddened*, and the effused fluid, by the presence of which the inflammation is, strangely enough, said to be established, is *white*—but there *can* be no inflammation *without* red globules.

Nay, I think that even an attentive consideration of *symptoms* might sometimes conduct us to the conclusion, that these have been falsely attributed to inflammation. Take the peritoneum; are not the *symptoms* recognised by authors as full evidence of its being *inflamed*, just the same symptoms which occasionally attend certain affections of *nerves*, as mentioned by Chomel. In certain neurotic diseases, he informs us, that exquisitely acute pain is felt, which comes on suddenly without any apparent cause, lasts from ten to twelve hours, *with nausea, vomiting, and quick pulse*, and yet gives way to the administration of opium. I remember a perplexity of this sort occurring in an hysteric patient, under my father's care in the Royal Infirmary of Glasgow, who, with the ordinary symptoms of hysteria, had, or as some then thought, affected to have, such extraordinary tenderness of the abdomen, that the smallest pressure made her cry out. I have now no doubt that the sensibility was real; she endured some sharp treatment for the removal of it, and it was only relieved with the relief of the general complaint. Dr. Elliotson, together with several other writers, has noticed similar cases where neurotic pain simulates so closely the pain of inflamed peritoneum as to render the diagnosis very far from easy.* Why, then,

* See Sydenham largely on the subject of Simulated Inflammation in Hysteric patients.

should not other exquisitely painful abdominal affections, if void of the constitutional symptoms of inflammation, be referred, as in these hysteric cases, to a neurotic cause? and why, in *either case*, should we refer pain to the *nerveless* peritoneum, and refuse the expression of it to the nervous structures in the vicinity? no serous membranes probably have nerves; for in health they are admitted on all hands to be *devoid of sensibility*, whatever stimulus be employed. Could this insensibility exist if they had *nerves?* or does inflammation effect, among other wonders, the *casual production of nerves*, or, confer the capacity to feel without them? consider Pericarditis, "Dans *beaucoup* de cas de pericardite," says Chomel, "il n'y a *aucun douleur* dans la region du cœur, et ce signe si precieux dans les autres phlegmasies manquent souvent ici, aussi bien *que la sensation de chaleur* qui l'accompagne ordinairement." It must be a strange pathology that would require us to admit *an inflammation* without *any one of its four signs*, pain, redness, heat, or swelling! But these cases are called *latent* inflammations; and *now* we see what latent inflammations are, and how they are characterized. It is by latent *pain*, latent redness, latent heat, and inappreciable swelling!! The remarks here made respecting inflammation of the peritoneum and pericardium will of course be found equally applicable to other serous membranes, and should seem to justify the conclusion, generally, both from the absence of pain in so many cases of what are called serous inflammations, and the fact of no nerves having been detected in them, that pain is neither *essential* to the diseases and disorganizations of these tissues, nor the tissues themselves, in accurate language, the seats of inflammation.

(10.) As inflammation cannot exist without redness, so, I conceive, that the increased redness of a part, is generally, but not always, an approach to inflammation. *Rubefacients* excite it in a slight degree, and one would almost refer the phenomenon of *blushing* to an action of vessels similar in kind, inasmuch as not only increased heat

but frequently a sense of tingling, which is of the nature of pain) and a feeling of distention, are perceptible.

In thus attempting to separate the pathology of inflammation from that of affections with which it is often confounded, if on the one hand, I have been made to feel more intimately, for my own profit, the difficulty which has been constantly expressed by the best writers on medicine, in discussing this very important and elementary subject, I am sensible, on the other, how imperfect has been the execution of my purpose—to call the attention of juniors in the profession like myself, to one or two among the many loose and inaccurate statements, which first obtain a spurious currency from authority, and then pass without question, from familiarity.

THE END.

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